(NEW SERIES.) No. 47.

SCIENTIFIC MEMOIRS

BY

OFFICERS OF THE MEDICAL AND SANITARY DEPARTMENTS

OF THE

GOVERNMENT OF INDIA

Dysentery and Liver Abscess in Bombay

BEING THE REPORT OF AN ENQUIRY CARRIED OUT BY

MAJOR E. D. W. GREIG, M.D., D.Sc., I.M.S.,

AND

CAPTAIN R. T. WELLS, M.A., M.B., I.M.S.

Under the direction of the Director, Central Research Institute, Kasauli

ISSUED UNDER THE AUTHORITY OF THE GOVERNMENT OF INDIA BY THE
SANITARY COMMISSIONER WITH THE GOVERNMENT OF INDIA, SIMLA

CALCUTTA
SUPERINTENDENT GOVERNMENT PRINTING, INDIA
1911

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CONTENTS.

PART I.

PAGE.

I.—INTRODUCTION 1

II.—GENERAL RESULTS 2

III.—BACILLARY DYSENTERY 5

IV.—AMŒBIC DYSENTERY 7
- (*a*) Methods and Plan of Work 8
- (*b*) Seasonal Prevalence of Amœbæ 9
- (*c*) Characters of Amœbæ 13
- (*d*) Amœbæ in Water 19
- (*e*) Distribution of Amœbæ in Healthy Animals 20
- (*f*) Experimental Observations on Animals 23
- (*g*) Experiments with Monkeys 23

V.—CLINICAL STUDY OF AMŒBIC DYSENTERY AND LIVER ABSCESS 29

VI.—DIAGNOSIS OF AMŒBIC INFECTION 49

VII.—GENERAL CONCLUSIONS 52

PART II.

1. TABLE I SHOWING RESULTS OBTAINED BY OBSERVATIONS ON CASES OF BACILLARY DYSENTERY . 55
2. TABLE II SHOWING VARIOUS SOURCES AT BOMBAY FROM WHICH AMŒBÆ WERE CULTIVATED . 64
3. TABLE III SHOWING THE RESULTS OF THE EXAMINATION OF THE STOOLS OF A NUMBER OF HEALTHY MONKEYS FOR AMŒBÆ 65
4. DESCRIPTION OF PLATES 77

ILLUSTRATIONS.

1. CHART NO. 1 SHOWING AMŒBIC PREVALENCE IN DYSENTERY CASES AMONGST THE CIVIL POPULATION AND THE TEMPERATURE CURVE 9
2. CHART NO. 2 SHOWING AMŒBIC PREVALENCE IN DYSENTERY CASES AMONGST THE MILITARY POPULATION AND THE TEMPERATURE CURVE 10
3. CHART NO. 3 SHOWING AMŒBIC PREVALENCE IN DYSENTERY CASES AMONGST THE CIVIL POPULATION AND THE HUMIDITY CURVE 10
4. CHART NO. 4 SHOWING AMŒBIC PREVALENCE IN DYSENTERY CASES AMONGST THE MILITARY POPULATION AND THE HUMIDITY CURVE 11
5. CHART NO. 5 SHOWING THE MONTHLY VARIATIONS FROM THE MEAN OF THE ADMISSIONS FOR DYSENTERY INTO THE JAILS OF BOMBAY 12

6. Text figure No. 1 showing life cycle of amœbæ 16
7. Chart No. 6 showing leucocyte and temperature curves of case No. 215 37
8. Chart No. 7 showing leucocyte and temperature curves of case No. 78 39
9. Chart No. 8 showing leucocyte and temperature curves of case No. 194 . . . 41
10. Chart No. 9 showing leucocyte and temperature curves of case No. 204 . . . 42
11. Chart No. 10 showing leucocyte and temperature curves of case No. 85 . . . 44
12. Chart No. 11 showing leucocyte and temperature curves of case No. 208 . . . 46
13. Chart No. 12 showing leucocyte and temperature curves of case No. 216 . . . 48
14. Plate No. 1 77
15. Plate No. 2 77
16. Plate No. 3 78

An Enquiry on Dysentery and Liver Abscess in Bombay.

PART I.

I.—Introductory.

IN December 1908 an enquiry into the causation of dysentery in India was undertaken by the Central Research Institute, Kasauli, under the direction of Lieutenant-Colonel D. Semple, M.D., Director of the Institute. Major E. D. W. Greig, M.D., I.M.S., and Captain R. T. Wells, I.M.S., were deputed from the Central Research Institute to carry out the investigation which was commenced at Bombay. Laboratory accommodation for the workers engaged in the enquiry was obtained at the Bombay Bacteriological Laboratory, Parel, and the Superintendent of the Laboratory very kindly gave every facility for the work to be carried out.

Cases of dysentery were examined in the Hospitals, both Civil and Military, in Bombay.

It became evident in the course of the enquiry that the distance of the Laboratory from the Hospitals (five miles) was a distinct disadvantage for the purpose of examining cases clinically and making *post mortem* examinations; accordingly it was decided that Captain Wells should devote his attention to the examination of the cases at the Hospitals and arrange to send the material for investigation to the Laboratory at Parel. Although a number of fatal cases occurred only a few *post mortem* examinations were permitted. Major Greig is responsible for the mode of presentation of the Report.

The plan of work included a detailed clinical examination of the cases on admission with notes of their subsequent progress, and the results of the *post mortem* examination when obtainable; the systematic working out of the material sent to the laboratory, namely, stools from dysentery cases, pus from liver abscesses, and blood for Widal reaction; a study of the various strains of

B. dysenteriæ isolated ; the investigation of amœbæ found in stools, liver pus, etc. The following methods were adopted for the examination of the stools :—(1) The reaction of the stool to litmus was tested. (2) A microscopic examination was made for the presence of amœbæ, ova of intestinal worms, etc. (3) A portion of the stool was plated on Drigalski-Conradi medium ; the composition of this medium, the method of plating, and examining the colonies have been fully described by one of us[1]. (4) The mucus was stroked, also, on plates of the agar medium of Musgrave and Clegg,[2] for the cultivation of amœbæ. The liver pus, which was collected in sterile test tubes, was examined microscopically for amœbæ, and culturally, both on ordinary agar, to determine whether or not it was sterile as regards bacteria, and on the agar medium of Musgrave and Clegg to obtain a growth of amœbæ. The temperature and humidity records for the year were obtained from the Meteorological Department, Bombay. During the investigations it became apparent that the number of cases of bacillary dysentery was small, and, consequently, in Bombay we were not afforded opportunities of studying the various problems connected with this type of dysentery. On the other hand a number of cases of dysentery associated with amœbæ in the stools came under our observation : therefore this variety of dysentery and its hepatic complications were studied in detail.

II.—General Results.

The following table shows the various Civil Hospitals attended in Bombay and the number of cases examined in each :—

Name of Hospitals.	Number of cases examined.
J. J. Hospital	117
G. T. Hospital	82
Northcote Police Hospital . . .	77
St. George's Hospital	105
Miscellaneous cases	16
TOTAL .	397

---

[1] Methods employed in the campaign against Typhoid.—Captain E. D. W. Greig.

[2] Bureau of Government Laboratory, Manilla, No. 18, October 1904.

The following table shows the various Military Hospitals in Bombay and the number of cases examined in each :—

Name of Hospital.	Number of cases examined
Colaba Station Hospital	46
116th Marathas ditto	70
109th Infantry ditto	49
Native details ditto	23
TOTAL .	188

The following table shows the total number of cases of dysentery examined amongst the Civil population and the presence or absence of amœbæ or dysentery bacilli in the stools :—

No. of cases of dysentery.		Amœbæ observed in	B. dysenteriæ observed in
Europeans . . .	121	47	2
Indians	248	114	4

The following table shows the total number of cases of dysentery examined amongst the Military population and the presence or absence of amœbæ and dysentery bacilli in the stools of each :—

No. of cases of dysentery.		Amœbæ observed in	B. dysenteriæ observed in
Europeans . . .	43	36	1
Indians	142	84	6

The following table shows the total number of cases of dysentery examined amongst the Civil population and the presence or absence of *Trichomonas* and the ova of intestinal worms in the stools :—

No. of cases examined.		Trichomonas.	Strongyloides.	Ova of worms.			
				Oxyuris.	Ascaris.	Trichocephalus.	Ankylostoma.
Europeans	121	13	1	0	2	2	1
Indians	248	38	6	3	22	12	7
Total .	369	51	7	3	24	14	8
Percentage . .		13·8	1·9	0·8	6·4	3·7	1·8

The following table shows the total number of cases of dysentery examined amongst the Military population and the presence or absence of *Trichomonas* and the ova of intestinal worms in the stools :—

No. of cases examined.		Trichomonas.	Strongyloides.	Ova of worms.			
				Oxyuris.	Ascaris.	Trichocephalus.	Ankylostoma.
Europeans	43	2	0	0	2	2	0
Indians	142	28	0	3	13	3	3
Total .	185	30	0	3	15	5	3
Percentage . .		16·2	..	1·6	8·1	2·7	1·6

The following table shows the reaction of the stool in the cases of dysentery tested amongst the Civil population :—

No. of cases examined.		Reaction of stool.	
		Acid.	Alkaline.
Europeans	59	19	40
Indians	122	68	54

The following table shows the reaction of the stool in the cases of dysentery tested amongst the Military population :—

No. of cases examined.		Reaction of stool.	
		Acid.	Alkaline.
Europeans	19	5	14
Indians	115	51	64

The following table shows the number of cases of liver abscess examined amongst the Civil population and the presence or absence of amœbæ in the pus and stools of each :—

No. of cases examined.		Amœbæ in	
		Pus.	Fæces.
Europeans	4	1	0
Indians	24	8	11

The following table shows the number of cases of liver abscess examined amongst the Military population and the presence or absence of amœbæ in the pus and stools of each :—

No. of cases examined.		Amœbæ in	
		Pus.	Fæces.
Europeans	3	1	0
Indians	0	0	0

III.—Bacillary Dysentery.

As already stated the number of cases of this condition, which were recognised bacteriologically in the course of our investigations at Bombay, was limited. The method employed for the detection of dysentery bacilli in the stools was as follows :—The mucus from a dysentery case was spread on three large plates of Drigalski-Conradi medium, which were allowed to dry, then set aside in the incubator at 37° for 24 hours[1]. They were examined carefully next day for suspicious colonies, which were tested with a high titre agglutinating polyvalent dysentery serum. If a colony reacted, it was sub-cultured on agar. From agar, tubes of glucose agar and litmus milk were inoculated in the first place, and if no gas was produced in the former, and in the latter the production of alkali, after a preliminary acid formation, without clotting, occurred, then it was grown in the various sugars. The indol reaction and the effect of inoculation into the rabbit was also determined. The morphological characters of the bacillus, motility, etc., as well as its behaviour towards Gram's stain, were noted in each case. In this way it was possible to determine exactly the variety of dysentery bacillus under observation.

---

[1] Methods employed in the campaign against Typhoid.—Captain E. D. W. Greig, I.M.S.

Each case of dysentery was examined a number of times for the *Bacillus dysenteriæ.* If positive, the examinations were continued into convalescence, after the stools had become feculent. In Appendix, Table I, the results of the investigation of each positive case are given. From a study of these results it will be observed that the excretion of bacilli in the stool is intermittent, as in the case of the *Bacillus typhosus.* When the blood and mucus ceased, and the stools became feculent, the presence of the *Bacillus dysenteriæ* was no longer detected, and for a time the patient would not be actively infective. There is not yet sufficient evidence to express a definite opinion as to whether, in a certain percentage of cases, the *Bacillus dysenteriæ* continues to exist in incompletely healed intestinal ulcers for long periods, to reappear in the stools again when the ulcers become active. The cultivation of the blood in cases of bacillary dysentery has not shown the presence of the *Bacillus dysenteriæ*, so that it is improbable that a certain number of dysentery cases can continue to harbour the bacillus in the bile and biliary passages as occurs in enteric fever.

As the lower part of the large intestine is frequently involved in bacillary dysentery the diagnosis might be aided considerably by an examination with the sigmoidoscope, a swab might be taken directly from the affected part for culture. It is possible that when bacteriological examination of the stool failed to demonstrate the presence of the *Bacillus dysenteriæ*, the nature of the case might be determined by cultivating the material removed by the swab. Further, the characteristic naked eye appearance of the large gut in bacillary dysentery should be revealed by means of the sigmoidoscope.

As regards the varieties of the *Bacillus dysenteriæ* it will be seen that out of the 13 cases in which it was determined 4 (30·8 per cent.) were produced by *B. dysenteriæ* (Shiga), and the remainder were caused by *B. dysenteriæ* (Flexner).

None of the cases of bacillary dysentery showed any indication of liver complication, either hepatitis or abscess. Nor did *B. dysenteriæ* occur in the stools of cases of liver abscess, although a careful and repeated examination was made.

It is of importance to note that, in the cases under observation, when the stools became feculent, the dysentery bacilli were not detected in them. It will be observed, also, that the excretion of the bacilli in some cases is intermittent. The full details of the results of the examination of each case of bacillary dysentery are given in Table I, Appendix.

The Widal reaction in bacillary dysentery was tested in a number of cases. It was not possible to separate an infection with *B. dysenteriæ* (Shiga) from an infection caused by *B. dysenteriæ* (Flexner) by this reaction owing to the group agglutination: as a method of diagnosis it is of less utility in dysentery than in enteric fever.

IV.—Amœbic Dysentery.

Whilst the bacillary dysentery has been studied by a number of competent observers, and an intelligent application of the principles founded on these investigations to particular cases will solve the problems connected with its prevention in most cases, on the other hand, when the subject of amœbic infection is approached, it is evident that a great many lacunæ in our knowledge still remain to be filled in. Our information regarding amœbæ at the present time may be compared to that of intestinal bacteria of some years ago. At that time the *B. coli communis* represented the so-called non-pathogenic intestinal organism and was compared with one standard type of pathogenic intestinal organism, the *B. typhosus*. At the present day the list of intestinal bacteria, which have been described, make a formidable array, and to the single pathogenic organism, the paratyphoid bacilli A, B and C and others must be added. This has been brought about mainly by the use of improved technique, but finality has not yet been reached. As regards amœbæ at the present time, most text books describe and contrast a pathogenic type (the *Entamœbæ histolytica*) and a non-pathogenic type (the *Entamœba coli*). Very recent work, however, has tended to extend these arbitrary limits and there can be little doubt that, as special technique improves, and further investigations are carried out, the number of amœbæ associated with dysentery will increase and the individuals in the group will be described more accurately.

There appears to have been a tendency in this field of investigation to adhere too rigidly to the conclusions arrived at by the eminent protozoologist, Schaudinn, although he himself admitted that lacunæ in our knowledge of the subject still remained to be filled in. Accordingly, it is interesting to note that Elmassian[1], in a recent paper describing a new species of pathogenic amœba, makes the following important remarks in this connection :—

> "Si les travaux de Schaudinn ont eu le grand mérite de mettre en lumière ce fait capital : la diversité des espèces amibiennes chez l'homme, ils en ont eu beaucoup moins d'après nous

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[1] Centralb. f. Bakt., etc., Orig. Bd. 52. Heft 3, 1909.

d'avoir crée cette fausse conception 'd'Amibe non pathogéne.' Il n'est pas certain que l'Ent. coli soit un parasite aussi inoffensif qu'on le croit. Et Gasser, quelque sceptique, qu'il puisse être sur le r le pathogène des amibes a tout de même constaté, 45 fois sur 109 cas de dysentérie, la presence de l'Ent. coli (cité par Patrick Manson). D'ailleurs a-t-on jusqu'ici examiné systematiquement les selles dans les cas de dysentérie nostras ? Est-on sûr que là on l'Ent. histolytica fût diagnostique il ne s'agissait pas par fois d'autres espèces ? Viereck[1] dit avoir trouvé son Amibe chez des personnes non dysentérique commes d'autres qui ont fait des observations analogues concernant les autres Amibes connues."

On this point Doflein[2] states "That Musgrave and Clegg have come to the conclusion from infections produced by amœbæ cultivated on artificial media that all free living amœbæ introduced into the intestine of animals can become pathogenic. While this is certainly going too far, I am of opinion that the question, whether many free living amœbæ may, in suitable culture, be transformed into pathogenic forms, is still open to discussion."

Koidzumi[3], also, described a species of amœba found in dysentery, which he believes is new to science, and calls it *Entamœba nipponica*.

(*a*) Methods and Plan of Work.

In approaching this difficult subject we followed certain definite lines. The fresh stool in each case of dysentery was examined microscopically for the presence or absence of amœbæ and a portion of the stool was stroked on the agar medium of Musgrave and Clegg, which had been poured previously into Petri plates and allowed to dry, and in this way cultures of amœbæ were obtained. It was found that the intestinal bacteria, ordinarily present in the stools, acted as symbiotic organisms, and it was not necessary to use other strains. The amœbæ in the cultures obtained were studied carefully in the fresh condition, and after fixation and staining. The pus from cases of liver abscess was treated in the same way. Cultures of amœbæ were made, also, from normal human fæces, sputum, collections of pus, the tap water of Bombay, and the stools (Appendix, Table II) and alimentary tract of various animals, *e.g.*, monkeys, rats, rabbits, guineapigs, etc. In this way we obtained a considerable amount of information regarding the morphology, life cycle, and distribution of amœbæ. The effects of feeding animals on cultures of amœbæ were noted also. Before proceeding to discuss the questions connected with the morphology and the life cycle of the amœbæ observed, and their relation to disease, it will be well to record certain broad facts regarding the seasonal prevalence of amœbæ and its relation to temperature and humidity.

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[1] Med. Klinik. 1906, No. 41.

[2] Lehrbuch der Protozœnkunde. Doflein 1909. P. 513.

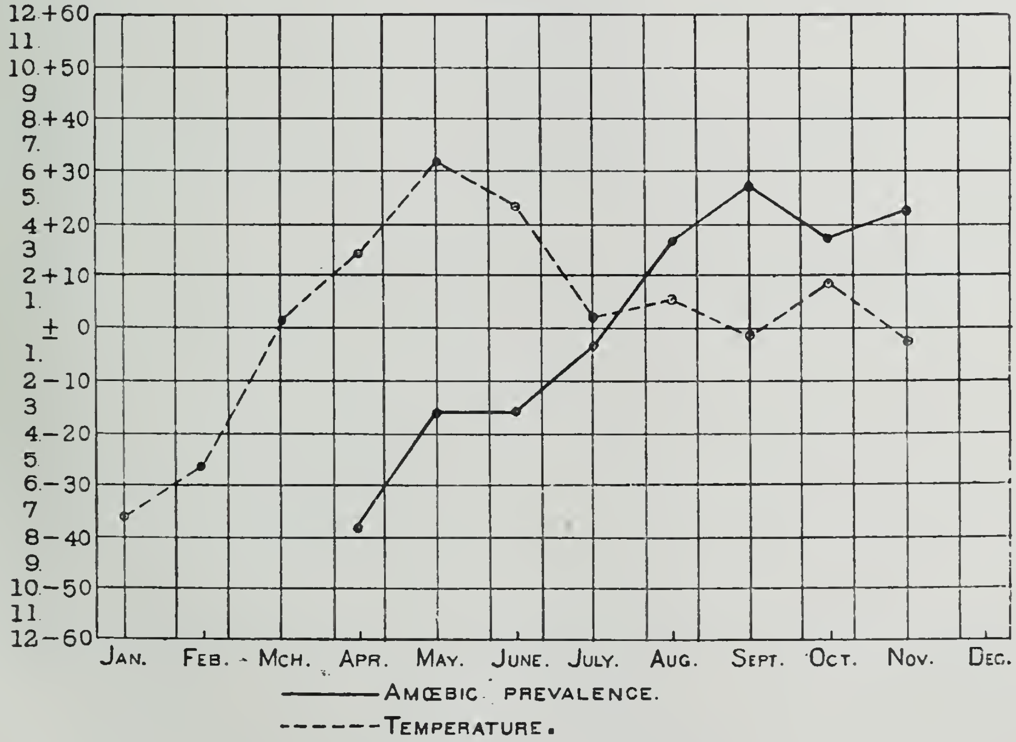
[3] Centralb. f. Bakt., etc., I Orig. Bd. 51 Heft. 6, 1909.

(*b*) Seasonal Prevalence of Amœbæ.

To determine this a portion of the stool of every fresh case of dysentery which came under observation was cultivated at room temperature on the agar medium of Musgrave and Clegg; this procedure was carried out daily for eight months, April to November 1909. The cultures were kept under observation and the presence or absence of amœbæ determined. In this way we were able to obtain the monthly incidence of amœbæ in dysenteric stools over three seasons in Bombay: (1) The hot weather, April to middle of June. (2) The monsoon, middle of June to end of September. (3) The period after the rains, October and November. Further, we obtained the daily records of temperature and humidity for the period under observation from the Meteorological Department, Bombay.

The following chart shows the monthly variations from the mean of amœbic infection in cases of dysentery examined amongst the Civil population in Bombay, and the monthly variation of temperature from the mean in Bombay :—

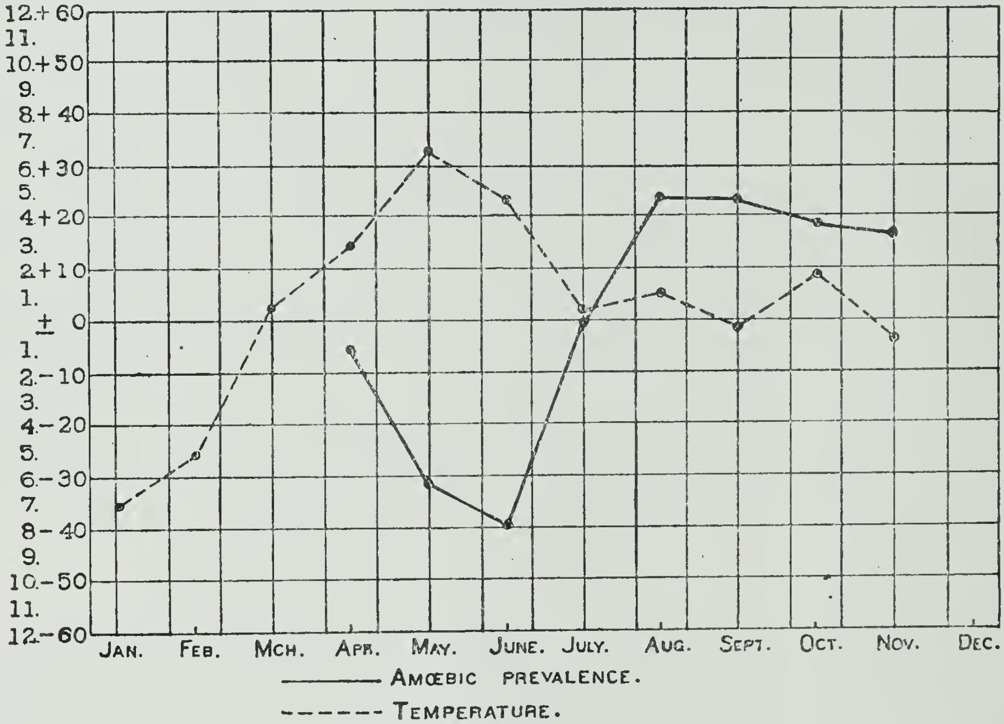
CHART I.



80·1 F. = Mean temperature.

The following chart shows the monthly variation from the mean of amœbic infection in cases of dysentery examined amongst the Military population in Bombay, and the monthly variation of temperature from the mean :—

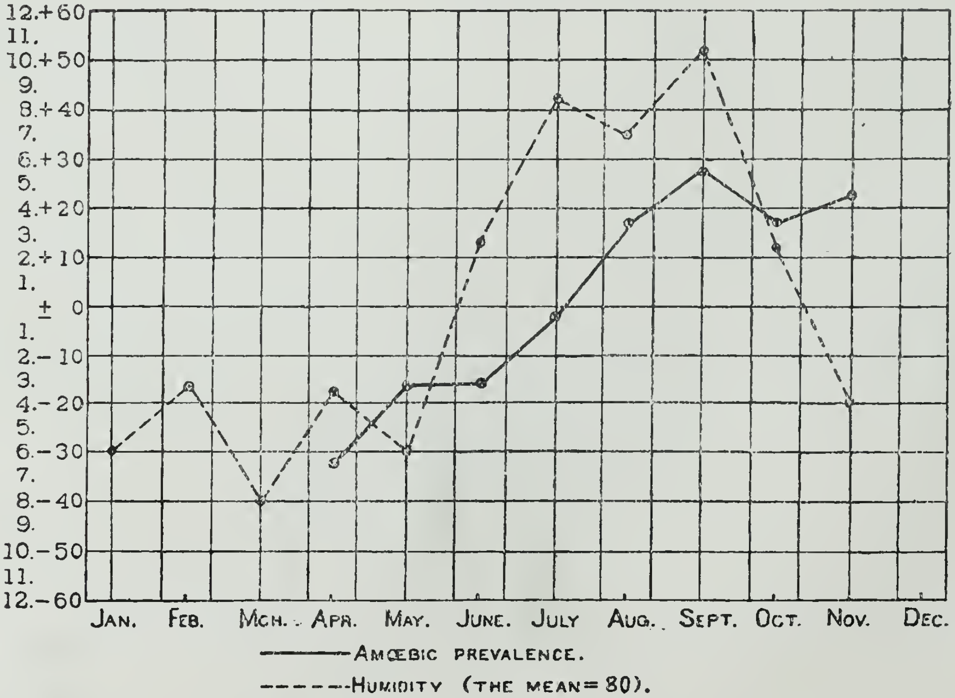
CHART II.



80·1 F.=Mean temperature.

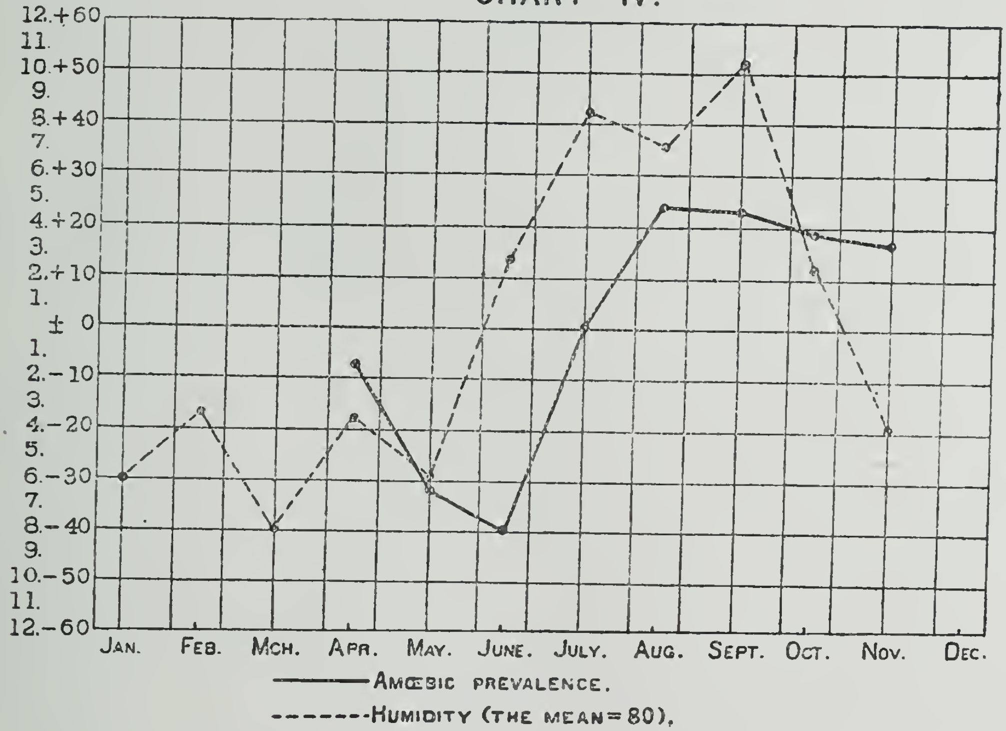
The following chart shows the monthly variation from the mean of amœbic infection in cases of dysentery amongst the Civil population in Bombay, and the monthly variation of humidity from the mean :—

CHART III.



The following chart shows the monthly variation from the mean of amœbic infection in cases of dysentery amongst the Military population in Bombay, and the monthly variation of humidity from the mean :—

CHART IV.



From a study of these charts it will be noted :—

(1) That amœbic infection shows a marked seasonal variation.

(2) That this variation is closely associated with the variations in humidity.

(3) That curves showing the variations in the amœbic infection and temperature do not correspond.

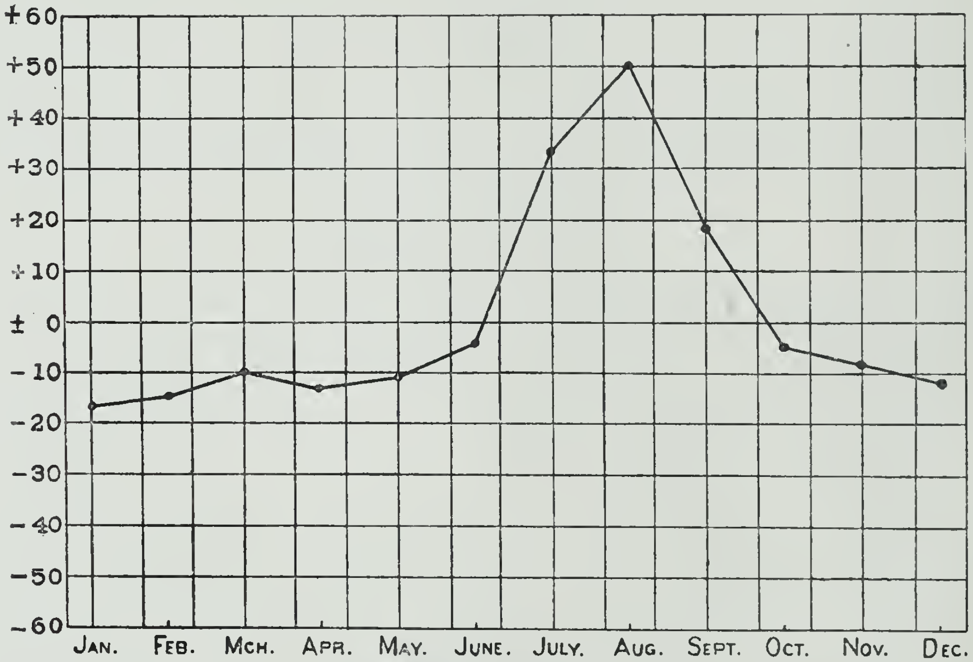
The intimate relationship of amœbic infection to moisture is significant, because moisture plays an important part in connection with the stage of encystment of amœbæ. If the medium in which the amœbæ are developing be allowed to dry, encystment is brought about readily. This stage in the life cycle of the amœbæ will be discussed later. In considering the seasonal prevalence of amœbic infection in cases of dysentery, all amœbæ, which could be cultivated from the stools on Musgrave and Clegg's medium, have been taken into account, not merely particular species, pathogenic or not. We have studied the natural conditions affecting the class as a whole. The peculiarities of individuals may form the subject for future investigation.

It is of interest and importance, further, to determine what relation the curve of amœbic infection bears to the curve of dysentery incidence in Bombay. To ascertain this the average admissions for dysentery to the Bombay Prisons for ten years, 1897-1906, were obtained for each month of the year from the Reports of the Sanitary Commissioner with the Government of India. These averages were added together and divided by the number of months, giving a mean admission rate for the period. The monthly variations from this mean were plotted out and a curve was obtained showing the seasonal variations of dysentery in the Bombay Prisons. On comparing this with the curve of amœbic infection it is noteworthy that there is a very close correspondence.

In both cases during the dry season the curve is below the mean, whilst in the wet season both are above. It indicates that the conditions necessary for the production of dysentery in the Jails in Bombay, as shown by the admission rate over a period of ten years, are favourable, also, to the life of amœbæ.

The following chart shows the monthly variations from the mean of the admissions for dysentery into the Jails of Bombay (1897-1906):—

CHART V.



ADMISSIONS FOR DYSENTERY.
BOMBAY PRISONS (1897-1906).
MEAN = 39.

The study of the intestinal fauna, especially of amœbæ, has received little or no attention so far. In the tropics, particularly, the subject would repay a careful investigation by competent observers and fresh ideas and conceptions might be expected to result from it.

(*c*) Characters of Amœba.

Having described the seasonal prevalence of amœbic infection in dysentery, we proceed to consider the results of the investigation of the amœba found. The amœba was studied in the stools and pus of liver abscesses, both fresh and stained. Further, amœbæ cultivated on the agar of Musgrave and Clegg from those and many other sources were carefully observed. We never were able to get the amœba to grow apart from bacteria. We obtained "pure mixed growths"; it would appear that, on artificial media, so far, bacteria are essential for the development of amœbæ, but that they can develop in certain conditions apart from bacteria is evidenced by their growth in the liver pus, which in most cases is quite sterile. It may be that certain strains of amœbæ can grow without bacteria; if this could be demonstrated much light might be thrown or the etiology of liver abscess. By growing amœbæ outside the body on artificial media a more complete study of the various phases in their life cycle can be made. A portion of the agar with a culture of amœbæ on it was placed on a slide, covered with a cover glass and ringed with vaseline; and in this way the amœbæ could be studied under the microscope in the fresh state for prolonged periods. Also, impression preparations were made from the agar culture, fixed wet in hot corrosive alcohol solution of Schaudinn, and stained by the iron hæmatoxylin method of Heidenhain; others were stained by Giemsa, but the best results were obtained by the iron hæmatoxylin method. Drawings, by means of a *camera lucida*, were made of a number of the preparations, fresh and stained. During our investigation the type of amœba met with agreed very closely with that described by Noc[1] in Cochin China, who worked out the life cycle in Mesnil's Laboratory at the Pasteur Institute, Paris. From the alimentary tract of healthy men and certain animals, amœbæ can be cultivated which present characters apparently similar to those obtained from the mucus of dysentery stools. Consequently, when monkeys are used for experimental feeding with encysted amœbæ, as was done by Musgrave and Clegg[2] and repeated by us, and symptoms of dysentery supervene with amœbæ in the stools, it is a matter of doubt as to which amœba caused the lesion, the one on which the monkey was fed or the amœba normally present. These facts indicate that the problem of the relationship of the various amœbæ to dysentery is a difficult and complicated one.

In this enquiry we have studied, as carefully as possible, the morphology, life history, etc., of the amœba cultivated from dysentery stools, liver pus, normal men and animals as well as tap water. Certain minute details connected with the unclear division and reproduction have been left for further study. Meantime cultures of amœbæ made from dysentery stools, liver pus, Bombay tap water, and the stools of normal monkeys were sent to Professor E. A. Minchin, F.R.S., University of London, and information was requested on the following points :—(1) If the four strains of amœbæ were the same. (2) Whether they were similar to the amœbæ described by Noc in Cochin China, but different from *Entamœba histolytica.* (3) As to their pathogenicity. Professor Minchin very kindly undertook to have the specimens examined ; and this was done by Miss M. Robertson M.A., Assistant Professor of Protozoology, London University, who has made a special study of amœbæ, and we are indebted to her for the following very interesting Report which she kindly sent us on the specimens :—

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[1] *Annal de L'Institute Pasteur*, No. 3, Mars 1909.

[2] *Ibid.*

> I have now looked through the slides from the amœba cultures. The amœba in the cultures from the dysentery patient, from the liver abscess and the Bombay water seem to me to belong to the same species, allowing that cultural forms give a true picture of specific characters.
>
> The amœba does not present the characters of either *Entamœba coli* or *Ent. histolytica ;* it seems, however, to be the same form as the one described by Noc from similar sources in Cochin China. The amœba in the culture from the monkey shows certain appearances not seen in any of the other three cases, although the trophic phase is identical. Whether these appearances denote a real difference of species or are a mere reaction to some cultural condition, I do not know, and it would take careful and prolonged study to determine this.
>
> Noc has well described all the more obvious features of the life-cycle and has stated a very neat case in favour of the form in question being the pathogenic agent in the periodic outbreaks of dysentery.
>
> There remain, however, many points in the cycle, points bearing more particularly upon nuclear features, multiplication, possible syngamy and autogamy which could be worked out from a close study of the cultures.

1. General Description.

The description of the amœbæ is based on the results of the direct examination of them in the stools and liver pus, and, also, on the study of the amœbæ in cultures from these sources. We were unable to determine any definite difference between the amœbæ cultivated from the above sources.

The protoplasm is divided into a granular endoplasm and a clear ectoplasm. There is a small excentrically placed nucleus at the boundary between the ectoplasm and endoplasm ; one or more contractile vacuoles are seen, also, (figures 6, 8, 9, 10, plate I). Contractile vacuoles are rare in parasitic amœbæ as a rule, but have been described by Noc[1] and Porter[2] in *A. chironomi.* Pseudopodia are formed during the movements of locomotion.

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[1] *Annal. de L' Institut Pasteur,* No. 3, March 1909.

[2] Journal of Parasitology, Volume II, 1909.

2. MOVEMENTS.

When seen in stools, pus, and in cultures, the amœbæ are actively motile as a rule. The movement is brought about by the throwing out of pseudopodia. Often the pseudopodium when first protruded is quite clear and hyaline, but the coarser granules from the body of the amœbæ rapidly stream into it.

3. MORPHOLOGY.

(1) *Size.*—This varies considerably according to the stage of growth of the animal as will be seen in Plates 1 and 2. The youngest amœba measures three to four microns (Figures 3 and 4, Plate I), the fully grown amœba being from thirty to forty microns in diameter, not including the pseudopodia (Figures 1 and 2, Plate I). It is important that this fact should be clearly realised, because in a stool or in pus from a liver abscess these small forms may predominate, and if the possibility of their presence is not appreciated a faulty diagonsis as regards amœbic infection may be given.

(2) *Endoplasm.*—This is generally coarsely granular, of greyish and sometimes greenish tint (Figure 7, Plate I).

(3) *Ectoplasm.*—In some cases it is difficult to observe a distinction, but when the pseudopodia are protruded the ectoplasm will be determined readily as a clear hyaline strip. It can be seen, also, in the stained preparation (Figure 7, Plate I).

(4) *Contractile vacuoles.*—These were observed in the amœbæ grown on agar, and, occasionally, in those in the stools. As a rule contractile vacuoles have not been recorded in parasitic amœbæ, but Noc[1] and Porter[2] have described them. They vary in number from one to three or more, they contract suddenly, once in 15—30 seconds. They appear to have a pinkish tint and are refractile. The number varies in the same amœbæ at different times.

(5) *Nucleus.*—This is excentrically placed at the boundary between the endoplasm and ectoplasm and is rich in chromatine which stains deeply, and which is sometimes seen to be surrounded by a clear unstained zone. The changes undergone by the nucleus, which are extremely important, will be described below. At certain stages a clear space is seen in the centre of the chromatine and a nucleolus may be observed also (Figures 1, 2, Plate I).

(6) *Encystment and reproduction.*—These processes require to be studied with great care as they form a method of classifying the various amœbæ. Schaudinn is of opinion that the study of the life-cycle of pathogenic protozoa should precede all experimental researches. To render clear the various

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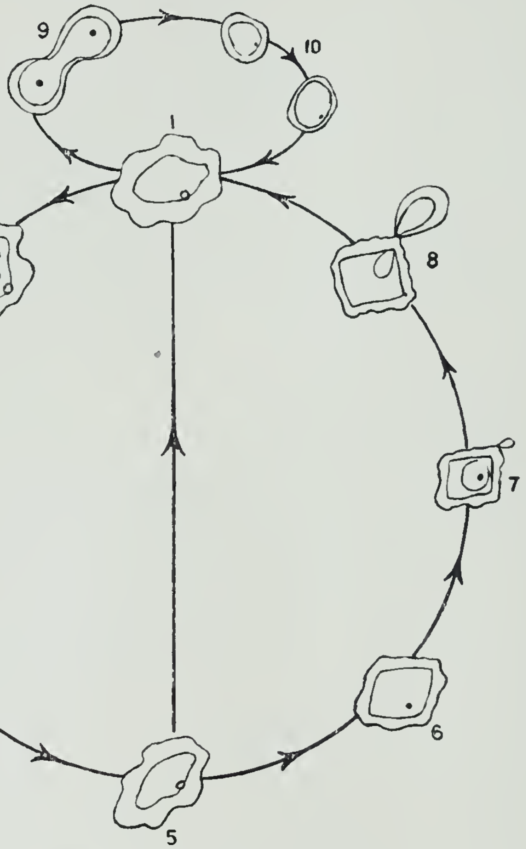
[1] *Annal. de L'Institut Pasteur,* No. 3, Mars 1909.

[2] Journal of Parasitology, Vol. II, 1909.

stages of the life-cycle of the amœbæ now being described the following diagram is given:—

DIAGRAM.
OF
LIFE CYCLE OF AMŒBA.



1—5 = Reproduction by budding.
6—8 = Stage of encystment.
9—10 = Reproduction by equal division.

If a culture of amœbæ of 24 to 48 hours growth be examined, the presence of a number of cysts will be observed. The cysts vary in shape, some are quite round, others are octohedral. They have a well marked double contour wall (Figures 13, 14, 15, 19, Plate I). In the octohedral form the outer part of the wall appears to be folded (Figure 15, Plate I), as if the inner wall had shrunk. In size they vary from 7 to 12 microns in diameter. When the amœba is about to encyst, it becomes spherical and the outer part condenses, forming the cyst wall. When such a cyst is placed under favourable conditions an interesting series of changes may be observed (Figure 28, Plate II). At first an irregular slow to and fro streaming of the cyst contents is observed, later these movements become very active and a bud of protoplasm is protruded through the cyst wall at one point. This bud shows amœboid movements and rapidly increases in size; at the same time the contents shrink from the cyst wall and an outward streaming of granules occurs. When the amœba has escaped, the empty cyst 'case' remains, and a small aperture at the point of exit of the amœba is observed. The cyst 'case' appears to be composed of a chitinous material. The whole process as above described took about two hours, the cysts being kept under observation at room temperature all the time (Figure 28, Plate II).

4. PRACTICAL IMPORTANCE OF CYSTIC STAGE.

Apart from the scientific interest, the cystic stage has a considerable practical significance, because in the stools this phase is not associated, as a rule, with active symptoms such as would lead to the detection of the case. Such individuals harbouring cysts of amœbæ are "Carriers," and may be a means of propagating the disease. Hence the necessity for a careful examination for cysts of the stools of cases convalescent from amœbic dysentery. Further, as Noc[1] has pointed out, the cysts are a source of danger not only to the community, but to the individual, because they occasion the relapses so frequently met with in amœbic dysentery. When conditions of the bowel are favourable the cystic stage of the amœba ends and a phase of active reproduction begins, this latter stage is dependent on factors which will be explained later. Myriads of young amœbæ may be seen in the stool, which resembles a rapidly growing culture (Figure 21, Plate I). In such a case the relapse may be rapid and exceedingly grave and the lesions found *post mortem* are extensive and severe. Hence in the interests of the patient before declaring a case 'cured', a careful examination of his fæces should be made.

Having considered the phase of encystment we pass to consider a very important process in the life-cycle of amœbæ, namely:—

5. REPRODUCTION AND NUCLEAR CHANGES.

In the examination of certain amœbæ (Figures 1, 2, 3, 4, Plate I) the endoplasm is seen to contain a large number of particles of chromatine, each surrounded by a minute portion of protoplasm. This round corpuscle measures 3—7 microns in diameter. If observed in the fresh state the corpuscles may be seen to make their way to the periphery of the amœba aided by the movements of the ectoplasm, and finally separate from the parent and move away (Figures 3 and 4, Plate I).

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[1] *Annal. de L'Institut Pasteur*, No. 3, Mars 1909.

These ' buds,' which become young amœbæ, are, generally, more or less spherical in form and the contour is defined (Figures 3 and 4, Plate I). The process is precisely similar to that described by Schaudinn, with this important difference that, in the amœba found in India by us, the ' buds ' become young amœbæ, whilst in *E. histolytica* the ' buds ' develop a wall of double contour and become cysts. Schaudinn describes the process in *E. histolytica* thus : " Slowly, 2—3 hours, under the influence of active currents of endoplasm, many small buds are expelled ; arriving at the periphery of the amœba they are finally eliminated as spores of concentric structure 3—7 microns in diameter." In the case of the amœba which we are at present considering the whole animal becomes enclosed in a cyst (Figure 22, Plate II). In this way it is distinguished from *E. histolytica.* In addition to reproduction by budding the amœba may divide into two daughter amœbæ, and as seen in Figures 5 and 20, Plate I, one or other daughter amœba may reproduce at once by budding.

By a careful study of numerous preparations fixed wet with alcohol acetic sublimate solution, and stained by Heidenhain's iron hæmatoxylin method, it has been possible to observe the nuclear changes which take place in the abovementioned processes of reproduction. There is a clear space in the centre of the nucleus which varies in size, the chromatine being arranged in a circular manner round it (Figures 1, 3, 4, 7, Plate I). The karyosome is seen to undergo a reduction into fine chromidia. These chromidia pass through the fine nuclear membrane and become diffused in the substance of the endoplasm. The original karyosome seldom disappears completely, and in most cases a portion remains. The grains of chromatine are contained in the endoplasm in small spherical masses of protoplasm, and constitute the ' internal buds '; in some cases (Figure 23, Plate II) these ' internal buds ' are very numerous, filling up the whole parent, which appears to be made up of these, and Noc[1] has named them ' merozoits,' which indicates the result of a division without expressing an opinion on the nature of the bodies. This unequal division of the nucleus appears to be the most frequent but, in addition, division by constriction (Figure 5, Plate I) into two equal parts followed by division of the protoplasm has been observed. Two daughter amœbæ result from this division. Noc[1] has observed division by karyokinesis with the formation of a regular nuclear spindle with two poles of chromatine and many grains of chromatine round the equator. We have noted, also, this form of nuclear division (Figure 29, Plate II), but division by amitosis appears to be more common than by mitosis. Noc also considers that the large number of chromidia emitted by a nucleus with a large karyosome explains, how in certain cases, the multiplication of amœbæ may be rapid and capable of producing more severe lesions than a simple bacterium ; the young amœbæ, issuing from the ' merozoits,' increase rapidly, and being young, nuclear division is favoured and premature buds are produced.

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[1] *Annal. de L'Institut Pasteur No. 3, 1909.*

An examination of cysts, fixed and stained by various methods, shows that the endoplasm of the amœbæ contained in them may be rich in chromidia, and, consequently, as soon as the amœba escapes from the cyst, it begins to bud rapidly (Figure 22, Plate II). The cysts of the dysentery amœba of Manilla described by Musgrave and Clegg[1] measure 10—12 microns, likewise in the amœbæ investigated by Lesage[2]; hence these amœbæ cannot be the same as *E. histolytica.* Craig[3] has studied the budding in amœbæ. The spore which he figures free does not correspond conclusively with the buds still adherent to the amœbæ. Walker[4] refutes the conclusion of Schaudinn in regard to the emission of buds which transform into cysts. From the above statements it will be seen that some subsequent observers have not been able to confirm the results of Schaudinn in regard to spore formation in *E. histolytica.* He himself recognised the lacunæ in his description of the life-cycle of *E. histolytica* and these still await filling in by zoologists.

(*d*) **Amœbæ in Water.**

In addition to dysentery stools and liver pus, ordinary Bombay tap water was examined for amœbæ. The method adopted was to take 2-3 litres of tap water and add a small quantity of ordinary nutrient broth to it. This was set aside at room temperature for about a week. Then from the surface several loopfuls were taken and plated on the special agar medium, and in the course of a few days a growth of amœba was obtained, if amœbæ were present in the water. A study of the amœba grown from water shows that it presents apparently similar characters to the amœba obtained from the stools of dysentery cases and from the pus of a liver abscess, hence it is unnecessary to repeat the description already given. The detection of this amœba in drinking water is a matter of interest. Further minute protozoological study will be required before the identity of the water amœba with those of dysentery and liver abscess can be established. Noc[5] from his observations in Cochin China considers that the amœba found in water is identical with the amœba found by him in dysentery stools and pus from liver abscesses. The following table, taken from his paper, shows the presence or absence of amœbæ in the water of Cochin China month by month:—

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[1] Bureau of Government Laboratory, No. 18, Manilla, October 1904.

[2] Ann. Inst. Pasteur, January 1905, XIX and C. R. Acad. de Sci. 26, December 1904.

[3] Journal of Infect. Dis., June 1908.

[4] Journal of Med. Research, Vol. XVII, No. 4, February 1908.

[5] *Ibid.*

Month.		Wells exposed to soilage.	Town water supply.
January		Cysts of amœba few	No amœba.
February		Ditto fewer	Ditto.
March		None	Ditto.
April		None	Ditto.
May	Period of excerbation of epidemic dysentery.	Cysts present	Cysts and motile amœbæ.
June		Cysts numerous	Abundant cult of amœba.
July		Ditto	Ditto.
August		Ditto	Ditto.
September		Ditto	Ditto.
October		Cysts less numerous	Ditto.
November		Cysts few	Cysts few.
December		....	None.

It is interesting to compare this table with the charts Nos. 1, 2, 3, 4, 5, pages 14—16, showing the incidence of amœbic infection and dysentery in man in Bombay. It will be seen that the season of the year during which the amœbæ are most numerous in the water of Cochin China and amœbic dysentery becomes epidemic, is the rains, and exactly corresponds to the period of greatest amœbic infection and highest dysentery incidence in man in Bombay. Therefore, in the present state of our knowledge, until further protozoological work on amœbæ is carried out in the East by trained protozoologists, and until such investigations conclusively show that the amœbæ of dysentery are absolutely distinct from those found in water, it will be necessary, in the interests of public health, to regard water as an important channel of conveyance of infection. That contamination of various foods may be brought about also by water is obvious, and therefore the taking of raw articles of food in endemic areas in India is not free from danger in regard to dysentery. It is of importance further in this relation to remember that the amœba forms cysts by which it is enabled to resist adverse conditions in nature, *e.g.*, absence of moisture, etc.

(*e*) Distribution of Amœbæ in Healthy Animals.

As amœbæ are found in drinking water it is obvious that they should be obtained from the contents of the alimentary tract of many animals. It is interesting to note in this connection that spontaneous amœbic dysentery, and also liver abscess in animals, have been noted by Kartulis,[1] Castellani[2] and ourselves. We have been able to cultivate amœbæ from the stools of different animals. The following table shows the kind and number of animals examined and the presence or absence of amœbæ in the stools :—

No.	Normal animal.	No. of normal animals.	Amœbæ in
1	Monkeys (Macacus sp.?) .	53	53
2	Kittens	6	6
3	Rabitts	47	43
4	Guinea pigs . . .	54	49
5	Rats	74	71
6	Flies, house (Musca domestica) contents of alimentary tract.	67	61

It was considered that a detailed study of the amœbæ found in normal animals did not come within the scope of the enquiry, hence no investigations have been made by us on their morphology and life-cycle. It is interesting to note that Miss Robertson inclines to the view that the amœba cultivated from the stools of the monkey differs somewhat from the amœba found in dysentery and tap water. We kept about 50 normal monkeys under observation, examining their stools at frequent intervals to determine the presence or absence of amœbæ and other parasites, as well as recording their weight and general condition during the period. In Table III, Appendix, the results of our investigations on the monkeys under observation, as well as a record of the monkeys which died with symptoms of dysentery, are given. It was important to make a careful study of normal monkeys in regard to the occurrence of amœbæ in their alimentary tract, because we were using these animals for feeding experiments, and they were used, also, by Musgrave and Clegg and Noc in their investigations on amœbic dysentery in Manilla and Cochin China respectively. It will be seen from a study of Table III, Appendix, that amœbæ were present in the stools of normal monkeys ; they were not detected every time the stools were examined, but at irregular intervals. This is a point of considerable importance, because if one or two examinations only had been made the monkey might have been regarded as free from amœbæ and used for experiment. The presence of parasitic amœba in the alimentary tube of healthy monkeys, which apparently closely resembles the amœba found in dysentery in man, introduces a serious experimental difficulty. The problem was complicated still further by the fact that, whilst under observation, some of the normal monkeys with amœbæ in their stools lose weight, develop symptoms of dysentery, and die, and the *post mortem* shows lesions in the large intestine.

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[1] Kolle und Wassermann, 1907.

[2] Journal of Parasitology, June 1907.

Hence it will be evident that the results obtained from feeding monkeys with amœbic cultures must be accepted with caution. The fact, however, that apparently healthy monkeys, in whose stools amœbæ have been determined, may develop symptoms of dysentery and die, is one of considerable interest and importance in connection with the etiology of the disease, although it does not establish conclusively the causal relationship between amœbæ and the symptoms.

In regard to the occurrence of spontaneous amœbic infection in animals an interesting observation was made by one of us (E. D. W. G.). A cat was being fed with a particular kind of rice for the purpose of an experiment not connected with the dysentery investigation. It showed no symptoms of dysentery during life, but became very much emaciated. It was unable to stand ; the hind legs were paralysed. It died on 26th February 1910. *Post mortem,* the stomach showed "patches" in several places, especially towards the duodenum and on the lesser curvature. These "patches" were hæmorrhages under the mucus membrane. In some places actual erosion of a superficial nature had taken place. The affected portions of the stomach were prepared, embedded in paraffin and cut. The sections were stained with methylene blue and eosine. On examining the sections an interesting appearance was noted (*vide* Plate III, Fig. 1) ; at one part of the section blood pigment was observed in the submucosa, near to this the mucous membrane was depressed, and in the epithelial layer a number of structures, taking the eosine stain strongly, were observed (Plate III, Fig. 2). Under a high power it was noted that the eosine staining bodies had the following structure : (1) a number of circular bodies varying in size and staining readily with the eosine ; (2) a blue stained body, the nucleus, with a central chromatine mass. The nucleus is always placed superficially to the circular bodies ; (3) 2 and 3 lie in clear hyaline material. Specimens were sent to Professor E. A. Minchin, F.R.S., for opinion, and he kindly examined them and gave the following very interesting Report :—"My impression of these bodies is that they are amœbæ of the histolytica type and that the eosinophil bodies are red corpuscles in various stages of digestion and absorption. Jürgens figures exactly similar appearances in dysenteric amœbæ, stuffed out with red blood corpuscles greatly diminished in size. The chief objection to regarding them as dysenteric amœbæ is their occurrence in the stomach. I never heard of them anywhere forward of the large intestine, but Jürgens infected his cats per rectum ; possibly if infected per os the amœbæ might start in the stomach. On the other hand the two chief characteristics of the histolytica type are the superficial nucleus, placed at the boundary between ectoplasm and endoplasm, and the greatly developed ectoplasmic layer: both these features are seen. The blood corpuscles in process of digestion would be in the endoplasm. I think therefore your cat had got an infection of amœba histolytica or some closely allied form. If they are not that, I don't know what they are. I certainly do not think they are any normal eosinophil cells of the animal."

(*f*) Experimental Observations on Animals.

The following table shows the results of experiments on cats and dogs with stools from dysentery cases and pus from liver abscess in which the presence of amœbæ were determined by microscopic examination :—

Serial No.	Material used and source.	Amœba.	Animal.	Mode of injection.	Result.
1	Mucus from dysentery case .	Present	Kitten.	Intrarectal .	Animal killed after two days — intestine healthy.
2	Scrapings from an ulcerated cæcum of fatal case of dysentery.	Do.	Pup .	Ditto .	Killed 15 days later—intestine healthy.
3	Scrapings from liver abscess wall.	Do.	Do.	Ditto .	Killed 20 days later—intestine and liver healthy.
4	Mucus from dysentery case .	Do.	Do.	Ditto .	Remained healthy.
5	Mucus from dysentery case .	Do.	Do.	Ditto .	Ditto.
6	Mucus from dysentery case .	Do.	Do.	Ditto .	Ditto.
7	Mucus from dysentery case .	Do.	Do.	Ditto .	Ditto.
8	Mucus from dysentery case .	Do.	Do.	Ditto .	Ditto.
9	Mucus from dysentery case .	Do.	Do.	Subcutaneously	Died two days later; much discoloration at site of injection. No amœbæ present.

From this table it will be seen that the experiments on cats and dogs gave negative results. Noc in his experiments on young cats with cultures of the amœbæ of Cochin China obtained negative results also. He considers this may be due to the acidity of the contents of the large intestine of these animals.

(*g*) Experiments with Monkeys.

Both Musgrave and Clegg in Manilla and Noc in Cochin China made use of monkeys in their experimental studies on amœbæ. Whilst Musgrave and Clegg, on the one hand, claim to have produced true amœbic infections in monkeys by feeding them on cultures, Noc, on the other hand, who has not succeeded in producing definite lesions in monkeys by feeding them with cultures of amœbæ, points out that dysenteric symptoms are not rare amongst monkeys, and adds that the bloody mucus, being a medium rich in red blood corpuscles and serous fluid, the amœbæ normally present develop in it. He is inclined to consider that the true cause of the dysentery in monkeys is not amœbic. In conclusion he asks the question, whether in the investigation of Musgrave and Clegg there was a true primary amœbic infection in monkeys at all. As we have already pointed out, amœbæ resembling those cultured from dysenteric stools of man can be cultivated from normal monkeys, and further, that these animals suffer from symptoms of dysentery which in some cases end fatally.

Hence in using monkeys for the production of experimental amœbic dysentery it is necessary to be aware of this fact, otherwise erroneous conclusions may be arrived at. We have carried out a number of feeding experiments in monkeys with cultures of amœbæ; details of some of these are given below. We have not been able to convince ourselves that we have induced a true amœbic dysentery in monkeys, and in this respect we are in agreement with Noc. Some of the animals have died with ulceration of the large intestine, which may have been of amœbic origin, but the fact that uninfected monkeys with amœbæ in their stools may die and show alterations in the large intestine renders the experimental results difficult of interpretation. From what has been said above it will be readily understood how complicated the whole subject is, rendering conclusive experimental proof of the pathogenicity of different species of amœba very difficult. In this connection, however, the quotation from Doflein's work (p. 8 of this Report) is of interest.

The following is an account of some of the feeding experiments carried out on monkeys :—

Ex. 7.—Monkey.

The strain of amœba used in this experiment was cultivated from the stools of a case of dysentery No. 8250 Private A, on May 10, 1909.

May 13th, 1909.—Two small petri plates of this strain, which showed many cysts, were emulsified with 0·75 NaCl, which had been rendered distinctly alkaline; each 100 c.c. contained 1 c.c. normal NaoH. This emulsion was introduced into the stomach of this monkey by means of a catheter. Weight of monkey 3,200 grams.

June 29th, 1909.—Two small plates 14th and 15th passage emulsified and given as before.

July 19th, 1909.—An emulsion of two plates 20th and 21st passage of the same strain of amœba showing many cysts was made in the same way and introduced into the stomach of this monkey.

August 2nd.—Since July 28th this monkey has had frequent small stools of blood and mucus : died this morning. *Post mortem.*—Weight 2,760 grams. On opening the abdomen patches of deep injection were visible in the large intestine, most marked in the transverse colon, sigmoid ard rectum. The omentum is firmly adherent to the transverse colon. Slight diffuse injection of the small intestine up to the duodenum. On opening the large intestine extensive ulceration is found ; the ulcers are very irregular, undermining the mucus membrane. *Small intestine.*—Slight congestion, no ulceration. No worms in alimentary tract. *Liver* and other organs show no noteworthy change. No motile amœbæ could be seen in a scraping from the ulcer, but a culture gave an abundant growth of amœbæ.

The following table shows the presence or absence of amœbæ in the stools and the weight of the monkey :—

No. of Monkey.	Weight (grams).	Stools.	Amœba.	
			Microscope.	Cultivation.
7	3,200—13th May 1909 .	..	— 29th April 1909 .	— 13th May 1909.
	3,280—17th May 1909.	No diarrhœa . .	— 30th April 1909 . Trichomonas.	— 17th May 1909.
	3,300—24th May 1909 .	Ditto . .	— 7th May 1909 .	— 24th May 1909.
	3,420—31st May 1909 .	Ditto . .	— 17th May 1909 .	— 14th June 1909.
	3,400—7th June 1909 .	Ditto . .	— 24th May 1909 .	— 19th June 1909.
	3,390—14th June 1909.	Ditto . .	— 14th June 1909. .	— 28th June 1909.
	3,390—21st June 1909 .	Ditto . .	— 19th June 1909. .	— 5th July 1909.
	3,350—28th June 1909.	Ditto . .	— 28th June 1909 .	— 16th July 1909.
	3,400—5th July 1909 .	Ditto . .	— 5th July 1909 .	+ 28th July 1909.
	3,370—12th July 1909 .	Ditto . .	— 16th July 1909 . Trichomonas.	+ 29th July 1909.
	3,300—19th July 1909 .	Ditto . .	— 28th July 1909. Trichomonas.	
	3,230—26th July 1909 .	Ditto . .	— 29th July 1909. Trichomonas.	
		28th July 1909—Diarrhœa and blood mucus.		
		29th July 1909— Do.		
		30th July 1909— Do.		
	2,760—2nd August 1909, died.	31st July 1909— Do.		

REMARKS.—This experiment is a suggestive one. The ulceration of the large intestine of this monkey was well marked.

Ex. 8.—Monkey.

May 25th, 1909.—The strain of amœba used in this case was cultivated from the stools of a case of dysentery, a Hindu male, aged 35, on May 21st, 1909, the amœbæ having been previously determined in the stools by microscopic examination. An emulsion of the culture, which contained many cysts, was made with alkaline 0·75 NaCl, and this was introduced into the stomach of the monkey by a catheter.

June 7th, 1909.—The monkey passed blood in its stools.

August 24th, 1909.—6 c.c. of Tinct. opii were injected into the peritoneal cavity of this monkey.

September 2nd.—Animal died. *Post mortem.*—Body emaciated. No fluid in the peritoneal or pleural cavities. *Large intestine.*—Mucus membrane appears to be congested and necrosed in parts. Ulceration present about the middle of its course. *Liver* and other organs show no noteworthy change.

The following table shows the presence or absence of amœbæ in the stools and the weight of the monkey :—

No. of Monkey.	Weight (grams).	Stools.	Amœba.	
			Microscope.	Cultivation.
8	3,100—1st May 1909	..	— 29th April 1909	— 17th May 1909.
	3,180—10th May 1909	..	— 30th April 1909 Trichomonas.	— 20th May 1909.
	3,280—17th May 1909	..	— 7th May 1909	— 22nd May 1909.
	3,300—19th May 1909	..	— 17th May 1909	— 24th May 1909.
	3,350—22nd May 1909	..	— 19th May 1909	+ 7th June 1909.
	3,300—24th May 1909	..	— 22nd May 1909	— 14th June 1909.
	3,350—31st May 1909	No diarrhœa	— 24th May 1909 Trichocephalus.	— 21st June 1909.
	3,360—7th June 1909	Diarrhœa with bloody stool.	— 7th June 1909	— 28th June 1909.
		No diarrhœa	— 14th June 1909	— 5th July 1909.
		8th June 1909	— 21st June 1909	16th July 1909.
		9th June 1909	— 28th June 1909	— 10th August 1909.

No. of Monkey.	Weight (grams).	Stools.	Amœba.	
			Microscope.	Cultivation.
	3,400—14th June 1909	No diarrhœa	— 5th July 1909	+ 23rd August 1909.
	3,380—21st June 1909	Ditto	— 16th July 1909 Trichomonas.	+ 26th August 1909.
	3,370—28th June 1909	Ditto	— 29th July 1900.	
	3,320—5th July 1909	Ditto	— 10th August 1909.	
	3,250—12th July 1909	Ditto	— 23rd August 1909.	
	3,230—19th July 1909	Ditto	— 26th August 1909. Trichomonas.	
	3,160—26th July 1909	Ditto.		
	3,110—2nd August 1909	Ditto.		
	3,070—9th August 1909	Ditto.		
	3,030—16th August 1909	Ditto.		
	2,990—23rd August 1909	Ditto.		
	2,260—1st September 1909.	Ditto.		
	Died 2nd September 1909.			

By way of contrast we submit the histories of monkeys which never had been fed on amœbic cultures, amœbæ being present naturally in their stools.

No. 13.—MONKEY.

30th June 1909.—Amœbæ cultivated from the stools of this monkey.

1st July 1909.—Animal has diarrhœa and has been steadily losing weight.

27th July 1909.—Died. *Post mortem.*—Body very emaciated. No fluid in the peritoneal or pleural cavities. *Large intestine.*—Congested throughout its whole length. In the upper half numerous sub-mucus hæmorrhages. Raised areas of bluish gelatinous appearance running transversely. Just below the edge of the cæcal valve one of the raised areas has broken down and formed an ulcer. Mesenteric glands enlarged. No worms in the alimentary tract. *Liver* normal, other organs show no noteworthy change.

The following table shows the presence or absence of amœbæ in the stools, and the weight of the monkey :—

No. of Monkey.	Weight (grams).	Stools.	Amœba.	
			Microscope.	Cultivation.
13	1,650—26th May 1909	..	— 26th May 1909 Omit strongyloides.	— 26th May 1909. 16th June 1909.
	1,350—31st May 1909	No diarrhœa	— 16th June 1909	— 22nd June 1909.
	1,330—7th June 1909	Ditto	— 22nd June 1909	+ 30th June 1909.
	1,270—14th June 1909	Ditto	— 30th June 1909 Trichomonas.	+ 1st July 1909.
	1,310—21st June 1909	Ditto	— 1st July 1909 Trichomonas.	+ 6th July 1909.
	1,210—28th June 1909	Ditto	— 6th July 1909 Trichomonas.	+ 7th July 1909.
		1st July 1909—Diarrhœa; no blood.	— 7th July 1909 Trichomonas.	+ 19th July 1909.
	1,120—5th July 1909	No diarrhœa	— 19th July 1909. Trichomonas.	
	1,000—12th July 1909	6th July 1909—Diarrhœa; no blood.		
	920—17th July 1909	7th July 1909—Diarrhœa.		
	Died 20th July 1909	8th July 1909—Do.		
		10th July 1909—Do.		

No. 42.—Monkey.

17th August 1909.—Amœba cultivated from the stools of this monkey.

21st August 1909.—Monkey has diarrhœa.

26th August 1909.—Monkey has been losing weight, is now emaciated and very weak. Diarrhœa still present. Killed by chloroform. *Post mortem.*—Body much emaciated. No fluid in peritoneal or pleural cavities. *Large intestine.*—Whole mucus membrane diffusely congested : below the ileo-cæcal valve a patch of deep congestion with sub-mucus hæmorrhage : about the middle of the large intestine is a gelatinous protrusion, the size of a split pea. Mucus membrane in places is superficially necrosed, no actual ulceration, no worms in the alimentary tract. *Small intestine* appears healthy. *Liver* healthy ; other organs show no noteworthy change. Scraping from the necrosed portion of the mucus membrane of the large intestine shows no amœbæ microscopically : amœba recovered by cultivation.

The following table shows the presence or absence of amœbæ in the stools, and the weight of this monkey :—

No. of Monkey.	Weight (grams).	Stools.	Amœba.	
			Microscope.	Cultivation.
42	1,670—13th August 1909 .	No diarrhœa . .	— 17th August 1909 . Trichomonas.	+ 17th August 1909.
	1,640—16th August 1909 .	Ditto . .	— 21st August 1909 .	+ 21st August 1909.
	1,409—23rd August 1909 .	21·S	— 23rd August 1909 . Trichomonas.	+ 23rd August 1909.
	Died 26th August 1909 .	23·S		

REMARKS.—These two monkeys form excellent control observations to the two previous feeding experiments. In all four monkeys amœbæ were cultivated from the stools. In all four pathological change was observed in the large intestine on *post mortem* examination.

The need for caution in regard to drawing conclusions from the feeding experiments on monkeys with amœbic cultures is obvious. In Appendix, Table III, the records of our observations on a large number of healthy monkeys are given.

GENERAL RESUMÉ.

Before leaving the question of amœbæ in relation to the etiology of dysentery and liver abscess it may be well to summarise the results very briefly. In the first place our findings very closely follow those of Noc in Cochin China. The amœba cultivated by us from the stools of dysentery cases and pus from liver abscesses, although resembling the *Ent. histolytica* at one phase of its life-cycle, differs from it particularly in regard to cyst formation. *Ent. Coli* has not been cultivated by us. The establishment of the pathogenicity of amœba by animal experiment is difficult and the results are uncertain. A further detailed study of the life-cycle of parasitic amœbæ of man by trained protozoologists is required, as lacunæ exist in the description of the classical *Ent. histolytica.*

V.—Clinical Study of Amœbic Dysentery and Liver Abscess.

Whilst the etiology of amœbic dysentery was being investigated a careful clinical record was made of each case, which included repeated blood counts, the examination of the blood for parasites, the taking of temperature records, observing the effects of treatment, etc., etc. In this way important data were accumulated, and in this section the results of these researches are set forth.

(*a*) BLOOD CHANGES IN AMŒBIC DYSENTERY AND HEPATITIS.

Total and differential counts of the leucocytes were made in a number of cases, and this was repeated generally on several occasions throughout the disease. Thus it was possible to determine the leucocytic formula in amœbic dysentery—a question, as will be shown later, of great practical as well as scientific interest.

The total number of red blood corpuscles was determined also.

The following table shows the total number of white and red blood corpuscles per c.m.m. of blood, the percentage of the various white blood corpuscles, the presence or absence of malarial parasites in the blood, and the presence or absence of amœbæ in the stools in dysentery cases :—

Serial No.	Name.	Age.	Sex.	Race.	Occupation.	Number of days in hospital and result.	Temperature.	Date of examination.	R. B. C.	W. B. C.	PERCENTAGES.				Blood. Malarial paras.tc.	STOOLS.	
											P. N.	S. M.	L. M.	E.		Character.	Amœbæ.
1	A. K.	28	M	E	Sepoy, 109th Infy.	41 days R.	Normal .	27th March 1909			64	18·5	14	3·5	—		+ (M)
2	A.	23	M	E	Private, 1st Glosters	53 days R.	99·6 to 104 .	14th May 1909.		9,650					—		+ (C)
								21st May 1909.		8,080	63	30	6·5	0·5			
								25th May 1909.		11,400	71	24	3·5	1·5			
								29th May 1909.		12,000							
								2nd June 1909		11,960							
								22nd June 1909			67	26·5	3	3·5			
3	A. F.	29	M	E	Cook . . .	6 days . R.	Normal .	16th June 1909.	3,990,000	28,400	83	10·5	5	1·5	—		
								21st June 1909.		10,400	73	21	3	3			+ (C)
4	B. G.	40	M	H	Coolie . . .	7 days . D.	96 to 100 .	31st March 1909	4,540,000	12,000	80	8·5	10	0·5	—		+ (M & C)
5	B. T.	24	M	H	Constable . .	4 days . R.	Normal .	15th April 1909	4,740,000	8,300	57	28·5	4·5	10	—		+ (M & C)
6	B. S.	31	M	H	Do. . .	4 days . R.	Do. .	2nd May 1909.		10,080	53	28	17·5	1·5	—	Blood and mucus.	+ (C)
7	C.	20	M	E		53 days sick leave.	Normal to 100	17th May 1909.		35,240	86·5	10	2	1·5	—	Blood and mucus.	+ (C)
								18th May 1909.		33,850	81·5	12	5·5	1			
								20th May 1909.		32,400							
								22nd May 1909.		30,370							
								26th May 1909.		22,130							
								28th May 1909.		17,000							
								1st June 1909.		14,860	80	16	3	1			
								3rd June 1909.		13,480	79	16	5	0			
								5th June 1909.		14,400							
								7th June 1909.		15,920							
								9th June 1909.		14,500							
								13th June 1909.		10,000	57	37·5	3	2·5			
								17th June 1909.			67	29	2·5	1·5			
								19th June 1909.		15,640	51	43	5·5	0·5			

Serial No.	Name.	Age.	Sex.	Race.	Occupation.	Number of days in hospital and result.	Temperature.	Date of examination.	R. B. C.	W. B. C.	PERCENTAGES.				Blood. Malarial parasite.	STOOLS.	
											P. N.	S. M.	L. M.	E.		Character.	Amœbæ.
								21st June 1909.		10,400	55	42	2	1			
								23rd June 1909.		12,400	48·5	46·5	3	2			
								25th June 1909.		12,000	49·5	45·5	3·5	1·5			
								27th June 1909.		10,760	47	44	5·5	3·5			
						R.		1st July 1909.	3,625,000	9,640	47	45	5·5	2·5			
9	P.T.C.	26	M	E	2nd K. R. R. . .	58 days R.	Normal .	7th June 1909.	4,376,000	11,900	67	25	5	3	—	Mucus and little blood.	+ (C)
								18th June 1909.		11,120	60	36	2·5	1·5			
10	G. K.	25	M	H	Coolie . . .	14 days R.	97 to 101 .	14th April 1909.	3,280,000	7,100	65	15·5	19·5		—	Mucus and blood.	+ (C)
								16th April 1909.		5,600							
11	D. A.	28	M	Goa	Student . .	34 days R.	Fever .	4th May 1909.		15,200	50	31	17	2	—		
								6th May 1909.		22,190	52	31	15·5	1·5			
								8th May 1909.		24,680							
								10th May 1909.		27,670	62·5	26	11	0·5		Mucus .	+ (C)
								12th May 1909.		16,560	63	33	3	1			
								13th May 1909.		20,570	60·5	33	5·5	1			
								14th May 1909.	6,100,000	19,920							
								15th May 1909.		18,410	51	40	5	4			
								17th May 1909.		15,500	47·5	45·5	4	3			
								19th May 1909	5,620,000	16,050	58·5	34·6	4·5	2·5			
								23rd May 1909.		15,700							
								26th May 1909.		14,620							
								28th May 1909.		12,900							
								29th May 1909.		10,400							
								1st June 1909.		13,000							
								4th June 1909.		9,280							
12	G.	36	M	H	Servant . .	16 days R.	Normal to 99.	21st May 1909.		36,720	75	20	3	2	—	Mucus and blood.	+ (C)
								23rd May 1909.	6,416,000	26,140							

								25th May 1909.	6,920,000	20,700	61	34	3	2			
								28th May 1909.		17,280	63·5	31	4·5	1			
								1st June 1909.		15,200	59	37·5	3·5	0			
								4th June 1909.		13,200							
13	H. D.	23	M	M	Sepoy, 116th Mah.	8 days R.	Normal	16th April 1909.	4,560,000	6,700	84	8·5	7·5		—		+ (M & C)
14	H. A.	25	M	M	*Nil*	8 days R.	Do.	24th April 1909.		6,700	60	28	12	0	—	Mucus and blood.	+ (C)
15	J.	..	F	E	*Nil*	52 days R.	Do.	13th June 1909.	1,325,000	7,000	67	30	2·5	·5	—	Do.	+ (C)
								20th June 1909.		8,760	77	18	3	2			
								28th June 1909.	2,870,000	7,000							
16	K. M.	25	M	H	Hawker	9 days R.	97 to 99	15th March 1909	5,470,000	12,000	70·9	0·5	19·9	8·7	—	Do.	+ (C)
17	K. R.	22	M	H	Shoemaker	7 days R.	97 to 99·6	31st March 1909	5,540,000	17,000	85·5	4·5	10	0	—	Blood chiefly	+ (C)
18	L.	25	M	E	R. E.	62 days R.	102 to 103	17th April 1909.		10,720	66·5	24	7·5	2	—	Blood and mucus.	+ (C)
								24th April 1909.		12,540							
								11th May 1909.		12,250	67	23·5	6·5	3			
								21st May 1909.	4,020,000	13,460							
								15th June 1909.	3,325,000	15,800	67	26	6	1			
								16th January 1909.		11,700							
19	L. C.	42	M	Jap.		3 days D.	Continued fever.	17th May 1909.		10,640	73	23	3·5	0·5	—	Blood chiefly	+ (C)
								17th June 1909.		18,720	89	8	2	1			
20	M. S.	38	M	H	*Nil*	22 days improved.		23rd June 1903.	1,300,000	3,480	15·5	80	4·5	0	—	Loose, trace of blood.	+ (C)
								28th June 1909.		3,980	25	70	5	0			
								30th June 1909.		3,380	36	61	2·5	0·5			
								21st July 1909.		4,160	34·5	61·5	3	1			
21	P. K.	36	M	M	Sepoy, 116th Mah.	13 days R.	Normal	16th April 1909	4,400,000	8,600	64	30·5	5·0	0·5	—		
22	P. S.	18	M	H	Sepoy, 116th Mah.	12 days R.	Do.	17th April 1909	4,350,000	9,000	66	27·5	5	1·5	—	Mucus and blood.	+ (C)
23	P. Gr.	24	M	E	R. G. A.	42 days R.	97 to 100	20th April 1909		4,260	56	33	9	2	—	Mucus and blood.	
								5th May 1909.		5,840							
								10th May 1909.		4,580	56	37	7	0			

Serial No.	Name.	Age.	Sex.	Race.	Occupation.	Number of days in hospital and result.	Temperature.	Date of examination.	R. B. C.	W. B. C.	PERCENTAGES.				Blood. Malarial parasite.	STOOLS.	
											P. N.	S. M.	L. M.	E.		Character.	Amœbæ.
								21st May 1909 .	3,040,000	8,450							
24	J.	46	M	Goa	Cook . . .	R.	Normal .	23rd March 1909	5,460,000	11,860	80	14	6	0	—		+ (M & C)
25	R. G.	36	M	H	P. C. . . .	12 days R.	Do. . .	24th March 1909	5,270,000	9,200	70	4	22	4	—	Blood and mucus.	+ (M)
26	R. J.	36	M	E	Gym. Instructor .	10 days R.	Do. . .	6th February 1909.		12,000	57	33	9·5	0	—		+ (C)
27	S. A.	43	M	E	Horn dealer . .	11 days R.	Do. . .	3rd June 1909 .			68	25·5	3	0·5	—		+ (C)
								5th June 1909 .		14,160							
								11th June 1909		11,600							
28	W. Gr.	38	M	E	R. G. A. . .	25 days R.	Do. . .	21st April 1909		6,800	57	29	11	3	—		
29		40	M	H	Tailor . . .	. .	Do. . .	10th June 1909	1,730,000	7,000					—		+ (C)
									1,500,000	4,500							
30	S. M.	35	M	M	Milkman . .	19 days R.	Do. . .	29th May 1909		13,000							
								23rd May 1909	4,870,000	12,140							
								26th May 1909		10,320					—	Blood and mucus.	+ (C)
								1st June 1909 .		11,400							
								6th June 1909 .		10,920							
31	S.	34	M	E	Spinning work .	18 days R.	Do. . .	24th May 1909	4,125,000	8,000	58	35·5	6	0·5	—	Loose, trace of blood.	+ (C)

H=Hindu. M=Mahomedan. E=European. R=Recovered. D=Died. M & C=Microscope and Cultivation.

In considering these results the cases may be divided for convenience into three classes—(1) mild, (2) severe, (3) complicated. *The first class,* where the symptoms are not marked, the lesion being confined to a few small localised ulcers in the cæcum, may show no increase in the total number of the leucocytes (cases Nos. 5, 13, 28, 31). A case like this may not be recognised and treated as dysentery. This is a question of considerable importance and urgency both from the point of view of the community and the individual, because unrecognised cases such as these may form the foci of epidemics; further, an extension of the infection from the bowel may occur and produce the very grave complication of abscess of the liver. Hence the question of the diagnosis of amœbic dysentery in its earliest stage is one of great importance and will be dealt with in the next section. It is a common *post mortem* observation in cases of abscess of the liver to find on opening the large intestine a few small ulcers in the cæcum which had given rise to no symptoms of dysentery. It is interesting in this connection to note that we have been able to cultivate (page 51) amœbæ similar to those found in dysentery and abscess of the liver from the stools of cases of abscess of the liver, although no active symptoms of dysentery existed at the time. *In the second class* of cases the signs of dysentery are severe, indicating an involvement of the whole length of the large intestine, and *post mortem* evidence bears this observation out, for in such cases the entire length of the large intestine shows extensive ulceration. A microscopic examination of a section of the intestine shows the characteristic appearance of an amœbic infection, namely, a great thickening of the sub-mucus layer, which is the portion of the intestinal wall chiefly affected. In this thickened layer localised areas of cellular exudation may be seen, and at the wall of these small "abscesses" many amœbæ may be observed (Figure 3, Plate III). Amœbæ are seen also in the thickened sub-mucus layer which, apart from the above-mentioned foci, does not show much cellular infiltration. Except at the part actually ulcerated the layer of tubular glands shows comparatively little alteration. In this class of cases the lesions are very extensive. The blood count during life in these cases may show a high degree of leucocytosis, cases 7 and 12. It must, however, be remembered that some cases of this nature show little or no leucocytosis. However, that a high leucocytosis may occur in these conditions should not be lost sight of in making a differential diagnosis between dysentery and dysentery complicated by lesions beyond the large intestine, the most common and serious complication of this nature being that of amœbic infection of the liver, but others, *e.g.*, pericæcal abscess, acute peritonitis, etc., may occur also.

The third group of cases is a very important one since it includes the serious condition of liver abscess. As Rogers[1] has shown the early stage of invasion of the liver by the amœbæ, before suppuration has occurred, may be recognised by careful examination of the blood, and if the appropriate treatment is applied at this period of the infection the very grave complication of abscess of the liver may be prevented. Hence the extreme importance, as a preventive measure in these cases, of studying the blood changes which occur. In a case of amœbic dysentery with very slight symptoms, indicating a limited lesion in the large intestine, or in cases where there are no active symptoms of dysentery at the time the patient comes under observation, if it is found that there is a distinct, but not excessive increase of the total leucocytes, the normal relative proportion being maintained, as a rule, and the temperature is raised, then there is strong reason to suppose that the amœbic infection is no longer confined to the large intestine, but has extended to the liver. At this early stage there may or may not be definite physical signs indicating involvement of the liver apart from the leucocyte curve. Hence the urgent necessity of making a careful count, total and relative, of the white blood corpuscles in such cases, as this gives the earliest information regarding the invasion of the liver, a matter of great importance in treatment.

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[1] Medico-Chirurgical Trans., Vol. 90, 1908.

(*b*) THE EFFECTS OF ADMINISTRATION OF IPECACUANAHA.

The administration of ipecacuanha in amœbic dysentery and hepatitis is closely bound up with the study of the blood changes in these conditions, because the earliest phase of amœbic invasion of the liver, which is the most favourable for treatment with this drug, is detected only by a blood examination. When ipecacuanha is given at this early stage of hepatitis a reduction of the temperature and leucocytes with amelioration of the symptoms takes place. As these questions are of very considerable practical importance in the prevention and treatment of so serious and fatal a disease as liver abscess, the records of several cases bearing on it are given.

CASES ILLUSTRATING GROUP 3.

Case No. 215. L. D., age 40, Goanese cook.

May 17th, 1909.—Patient was admitted on 12th May complaining of pain in the right hypochondrum of five days duration. The onset was very sudden with severe pain in that region, nausea and vomiting. Patient felt feverish, weak, and ill.

Previous health.—Good, no history of dysentery.

Social condition.—Born in Mangalore ; has been in Bombay for sixteen years ; returned from leave from Goa ten months ago. Has habitually drunk country liquor, but does not admit excess.

Present condition.—Patient looks thin and debilitated, conjunctivæ have a faint yellow tinge. Tongue covered with a thick white fur. Abdomen is slightly retracted and moves freely with respiration, both iliac fossæ free from tenderness, *Colon* not palpable. *Spleen* not palpable. *Liver* very tender, enlarged, mostly downwards in front; area of dullness in middle line 3¾ inches, in nipple line 6¼ inches. Upper border of dullness cuts the mid line in the 6th interspace and the nipple line in the 5th interspace. *Lungs,* bronchitis râles at bases ; otherwise normal.

Liver was punctured in three places in front. A small quantity of sanguineous fluid only withdrawn, no pus.

May 31st, 1909.—Temperature normal, tongue clean. Liver mid line 3¼ inches. Right nipple 4 inches, upper border at 6th rib. No tenderness.

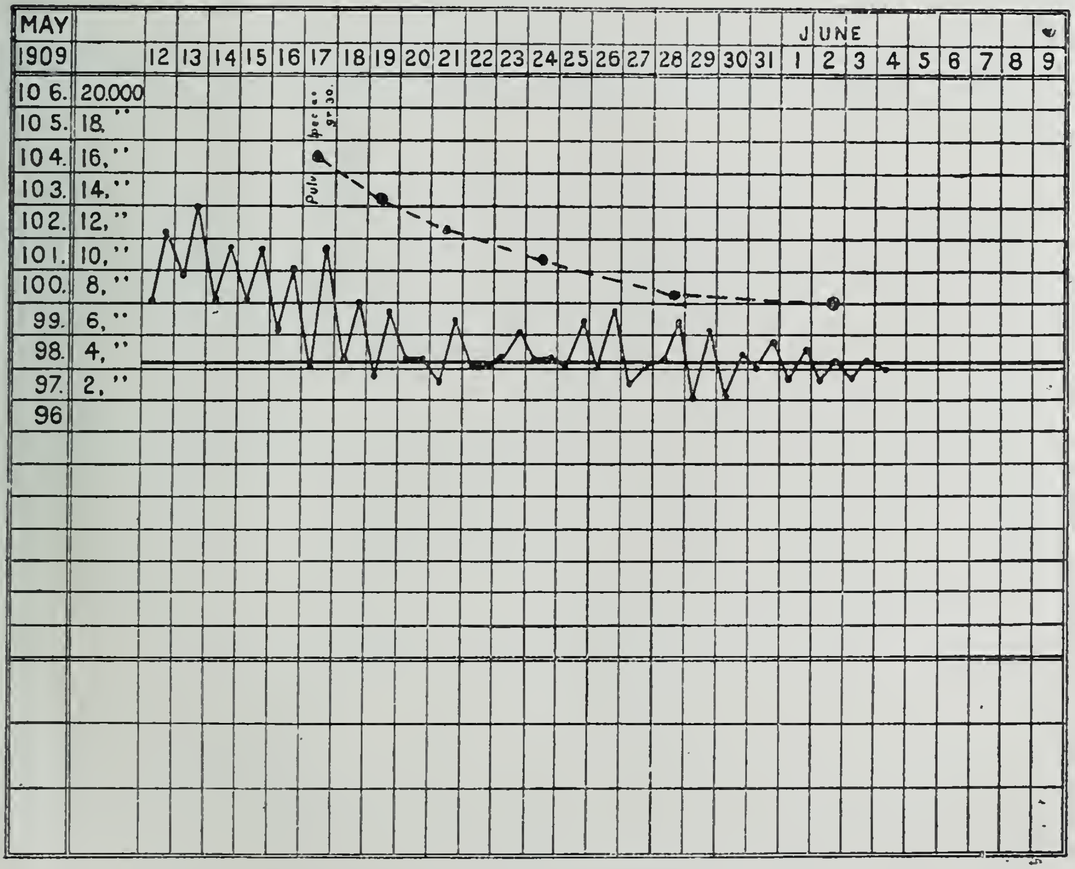
June 5th 1909.—Discharged.

Treatment—Pulv. ipecac, gr. 30, twice daily.

Cultivation of stools showed presence of amœbæ. An attempt was made to cultivate amœbæ from the sanguineous fluid withdrawn from the liver by puncture, but this failed.

The following chart shows the course of the disease and the total number of W. B. C. per c.m.m. of blood :—

CHART VI.



----TOTAL LEUCOCYTES. PER CMM OF BLOOD.
———TEMPERATURE.

The following table shows the result of the examination of the blood :—

Name and occupation.	Age.	Date of examination.	R. B. C.	W. B. C.	Percentages.				Parasites in blood—malaria.
					P. N.	S. M.	L. M.	E.	
Desouza, cook .	40	17th May 1909		16,880	57	36	4·5	2·5	—
		19th May 1909		14,050	62·5	32	4	1·5	
		21st May 1909	3,990,000	13,320					
		24th May 1909		11,550					
		28th May 1909		9,680					
		2nd June 1909		9,000	56	39	2·5	2·5	

REMARKS.—This case is an interesting and suggestive one. It illustrates several important points. In the first place the patient gave no definite history of dysentery ; this is not infrequent in cases of hepatitis and liver abscess, the reason being that the lesion of the bowel produced by the amœba is, at times, very slight and situated in the cæcum ; thus the symptoms produced are not urgent and no attention is paid to them, or they may escape notice entirely. The cultivation of the amœba from the stool in this case was of considerable interest. This patient suddenly developed fever with marked hepatitis and, also, some enlargement. There were no malarial parasites in the blood, but there was a distinct leucocytosis, the relative proportion of the leucocytes, however, remaining more or less unaltered. The most important practical point in the case was, that under full doses of ipecacuanha the fever and leucocytosis diminished, and finally all the symptoms cleared up and the patient was discharged well. There can be little doubt that this case was an example of amœbic hepatitis, which if it had not been recognised but had been left untreated might have passed on to abscess formation. Hence the importance of a careful blood examination and systematic treatment with ipecacuanha in such cases.

As a control observation the following case is given which is also one of hepatitis but in which ipecacuanha was not exhibited, another line of treatment being adopted.

Case No. 78. L., European, age 25.

April 17th, 1909.—Admitted to hospital complaining of abdominal pain with passage of blood-stained stools daily, tenesmus and straining. Left Rangoon on 9th, and shortly after noticed blood in the stools with loss of appetite, but was not severely ill.

Previous health.—Good, no dysentery.

Present condition.—Patient is somewhat pale and thin, but fairly well nourished. Tongue covered with brown fur. Abdomen normally full, no tenderness on pressure. Spleen not enlarged. Liver not enlarged.

April 24th, 1909.—Patient feels better. One or two semi-solid motions daily without griping ; no blood or mucus.

May 5th, 1909.—No diarrhœa ; no pain or tenderness.

May 21st, 1909.—No noteworthy change.

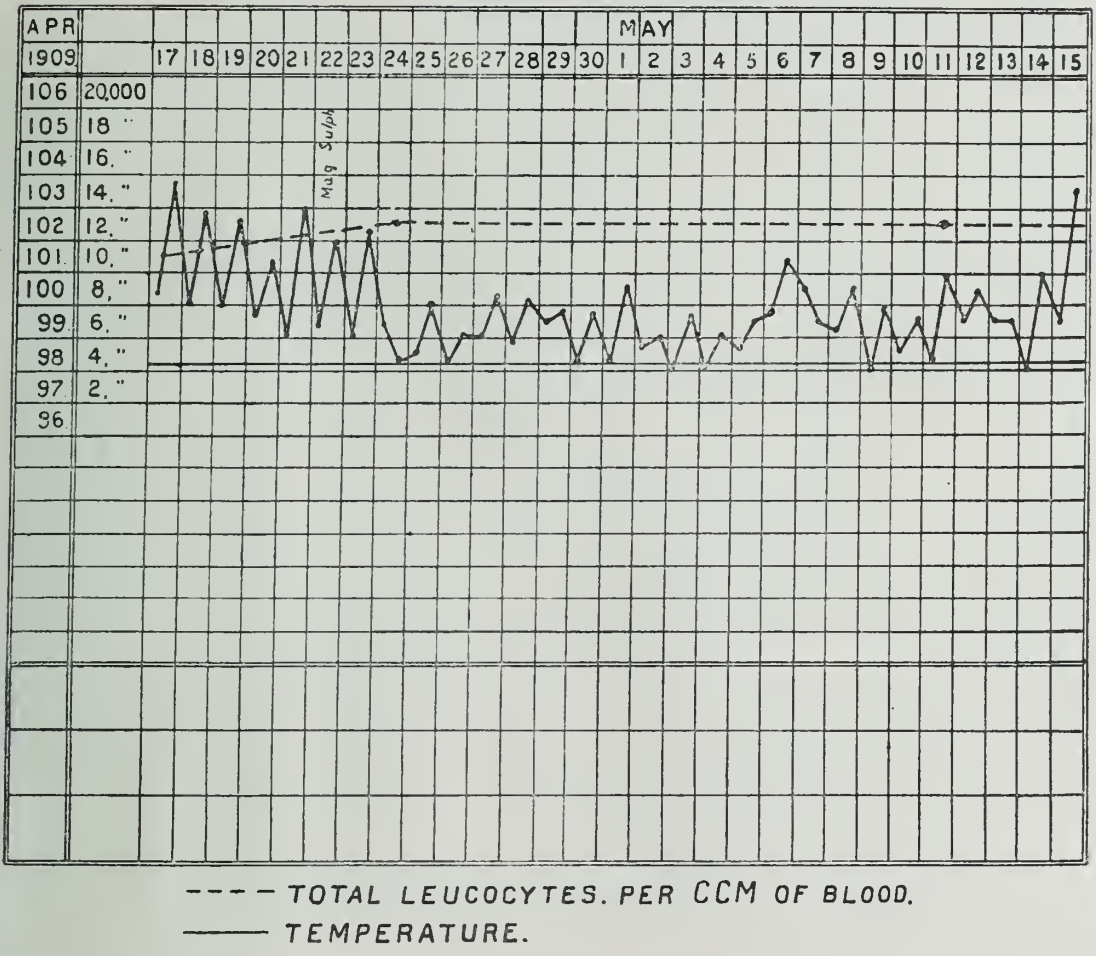
June 15th, 1909.—Bowels fairly regular. Motions healthy. Complains of pain in the right hypochondrium during the past week, now tending to shift upwards, towards the right shoulder. *Liver* enlarged downwards in front 1″ below costal margin in right nipple line ; upper border normal.

Examination of the stools showed the presence of amœbæ.

Treatment.—Mag. sulphate one drachm thrice daily and quinine.

The following chart shows the course of the disease and the total number of W. B. C. per c.m.m. of blood:—

CHART VII. —(*Continued, p. 40.*)



The following table shows the result of the examination of the blood:—

Name.	Age.	Date of examination.	R. B. C.	W. B. C.	PERCENTAGES.				Parasites in blood—malaria.
					P. N.	S. M.	L. M.	E.	
L. . .	25	17th April 1909 .		10,720	66·5	24	7·5	2	—
		24th April 1909 .		12,540					
		11th May 1909 .		12,250	67	23·5	6·5	3	
		21st May 1909 .	4,020,000	13,400					
		15th June 1909 .	3,325,000	15,800	67	26	6	1	
		16th June 1909 .		11,700					

REMARKS.—In this case we have the mild attack of dysentery, the presence of amœbæ in the stool. The symptoms of dysentery improve, but the temperature is irregular and there is a distinct leucocytosis. At first there are no hepatic symptoms, but these develop after he has been about two months in hospital. In this case we have a control observation to the previous one, the morbid condition was the same in both, but, in the former, ipecacuanha was exhibited with the result that the signs and symptoms disappeared; in the latter Mag. sulphate was used and the course of the disease was prolonged, and the signs and symptoms did not clear up. Such a case as this may ultimately develop liver abscess.*

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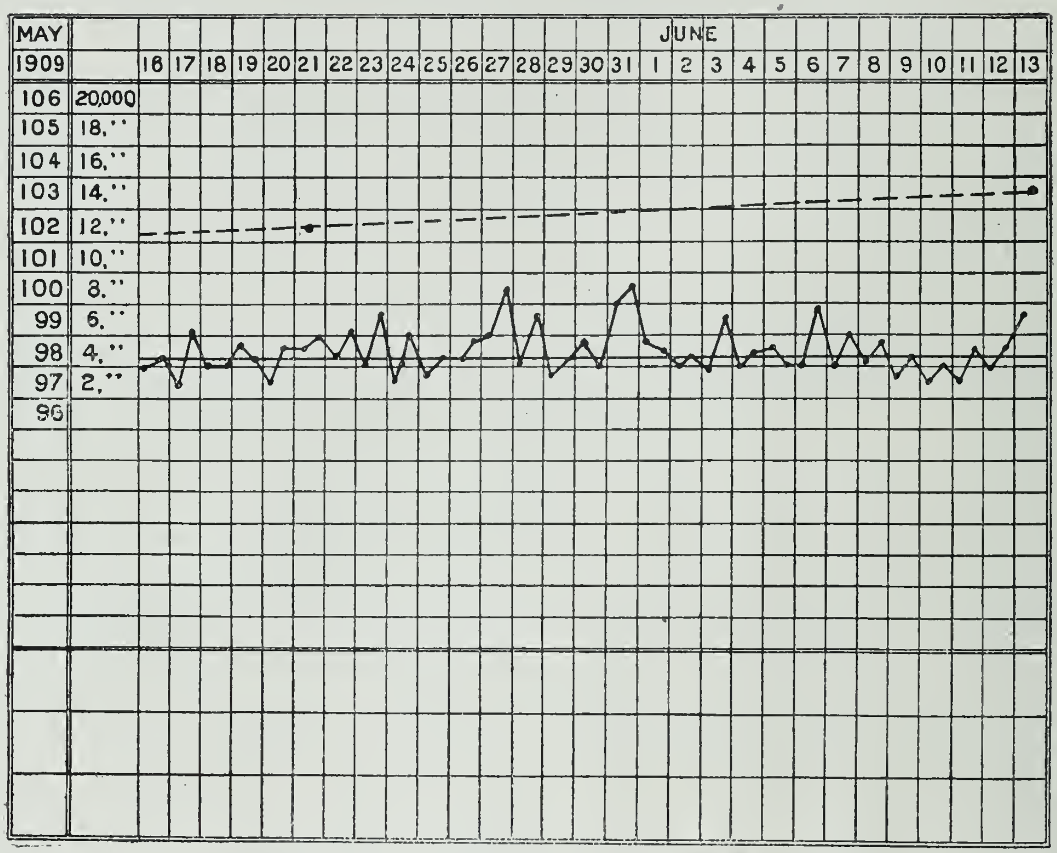
* It is interesting to record that this patient actually did develop liver abscess.

CHART. VII. (Cont).



- - - TOTAL LEUCOCYTES. PER CMM OF BLOOD.
——TEMPERATURE.

The two following cases are given to show the effect of administration of ipecacuanha on the temperature and leucocytosis in cases of dysentery :—

Case No. 194. B., European Seaman, age 33.

April 30th, 1909.—Admitted on 26th April complaining of griping pains in the abdomen with looseness of the bowels of a month's duration. Mucus and blood present in stools. Onset was gradual with ordinary diarrhœa.

Previous health.—Good ; no previous dysentery.

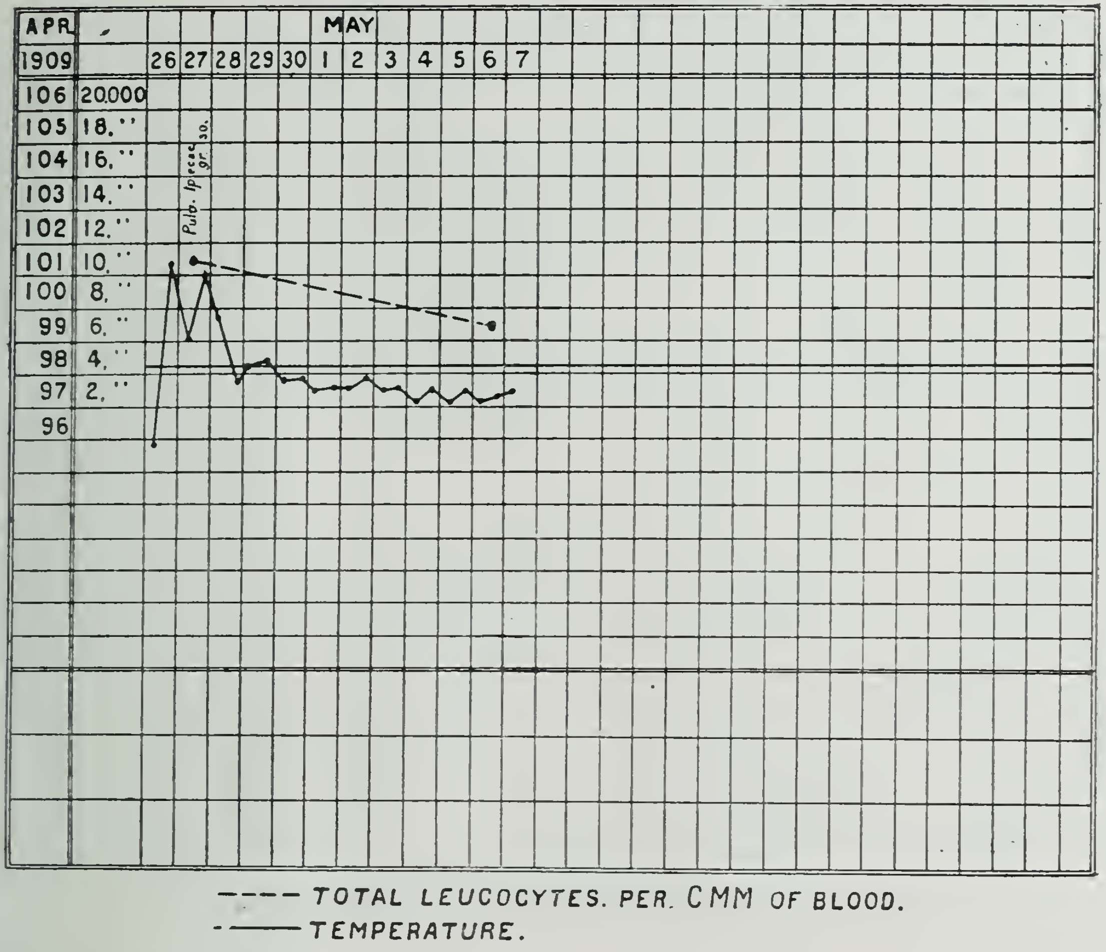
Social condition.—Born in India ; went home first in January 1909. Since then has done another trip to Falmouth. States that food on board was bad.

Present condition.—Patient fairly well nourished and well developed. Tongue covered with a yellowish white fur ; abdomen retracted. Slight tenderness on pressure in right iliac fossa. Spleen and liver not enlarged. Bowels now moved 2-3 times daily ; no straining and tenesmus.

May 7th, 1909.—Dismissed well. Treatment. Pulv. ipecac., gr. 30, twice daily. Amœba cultivated from stools.

The following chart shows the course of the disease and the total number of W. B. C. per c.m.m. of blood :—

CHART VIII.



The following table shows the result of the examination of the blood :—

Name and occupation.	Age.	Date of examination.	W. B. C.	Percentages.				Parasites in blood—malaria.
				P. N.	S. M.	L. M.	E.	
J., Seaman.	33	27th April 1909	11,650	64	30	5	1	—
		6th May 1909		56	35	7	2	

REMARKS.—This case shows the rapid fall of temperature and leucocytosis under ipecacuanha. Although there were no direct physical signs of acute hepatitis yet the occurrence of fever and leucocytosis in the mild case of amœbic dysentery pointed to a probable early invasion of the liver, and the action of the ipecacuanha confirmed this view.

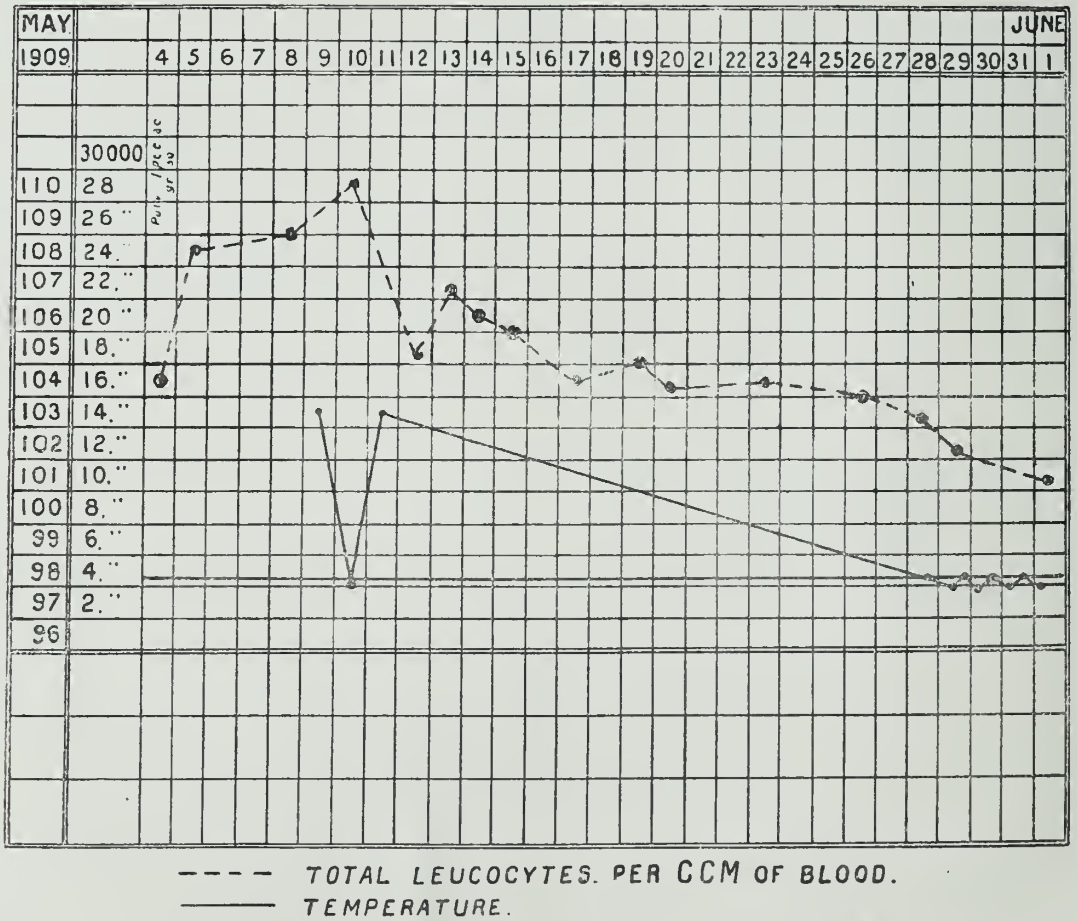
Case 204. A. D., Medical Student, age 28.

May 5th, 1909.—Admitted to hospital on 4th May complaining of pains in the abdomen with frequent motions, composed chiefly of mucus streaked with blood. Commenced about a week ago with diarrhœa without griping. No previous history of dysentery, no malaria.

Present condition.—Patient is well nourished and well developed; does not appear actully ill. Tongue covered with uniform white coating. Slight tenderness in right fossa. Spleen and liver not enlarged.

May 10th, 1909.—Tongue coated with brown glaze ; 4-5 stools in the day. No pain or tenderness in hepatic region. Temperature rose suddenly to 103·8 to-day, it was normal in the morning. Abdominal pain and tenderness very slight. Most tenderness in right iliac fossa, no marked local resistance. Liver and spleen not palpable or enlarged on percussion.

CHART IX



May 13th, 1909.—Tongue still coated with brown glaze in centre, but clearing.

May 15th, 1909.—General condition improving.

May 23rd, 1909.—Much improved. Temperature normal.

May 26th, 1909.—Improvement maintained, only 1-2 motions in 24 hours.

June 6th, 1909.—Tongue clean ; one solid motion in 24 hours.

June 7th, 1909.—Dismissed well. Treatment. Pulv. ipecac., gr. 30, twice daily.

The chart on page 42 shows the course of the disease and the total number of W. B. C. per c.m.m. of blood.

The following table shows result of the examination of the blood :—

Name.	Age.	Date of examination.	R. B. C.	W. B. C.	PERCENTAGES.				Parasites in blood—malaria.
					P. N.	S. M.	L. M.	L.	
D. A. . .	28	4th May 1909		15,200	50	31	17	2	
		6th May 1909		22,190	52	31	15·5	1·5	
		8th May 1909		24,680					
		10th May 1909		27,670	62·5	26	11	0·5	
		12th May 1909		16,560	63	33	3	1	
		13th May 1909		20,570	60·5	33	5·5	1	
		14th May 1909	6,100,000	19,920					
		15th May 1909		18,410	51	40	5	4	
		17th May 1909		15,500	47·5	45·5	4	3	
		19th May 1909	5,620,000	16,050	58·5	34·5	4·5	2·5	
		23rd May 1909		15,700					
		26th May 1909		14,620					
		28th May 1909		12,900					
		29th May 1909		10,400					
		1st June 1909		13,000					
		4th June 1909		9,280					

REMARKS.—In this case, also, during a moderately severe attack of amœbic dysentery leucocytosis developed and the temperature was elevated. There were no definite signs of enlargement of the liver, although the leucocytosis and fever disappeared under treatment with large doses of ipecacuanha.

The following illustrates the value of ipecacuanha in a case of liver abscess which had been opened and drained, and where it was found necessary to open a second abscess. The temperature and leucocyte count after the second operation still remained elevated, accordingly the patient was placed on large doses of ipecacuanha with the result that both the temperature and leucocyte curve fell to normal.

Case 85 T., European, age 28.

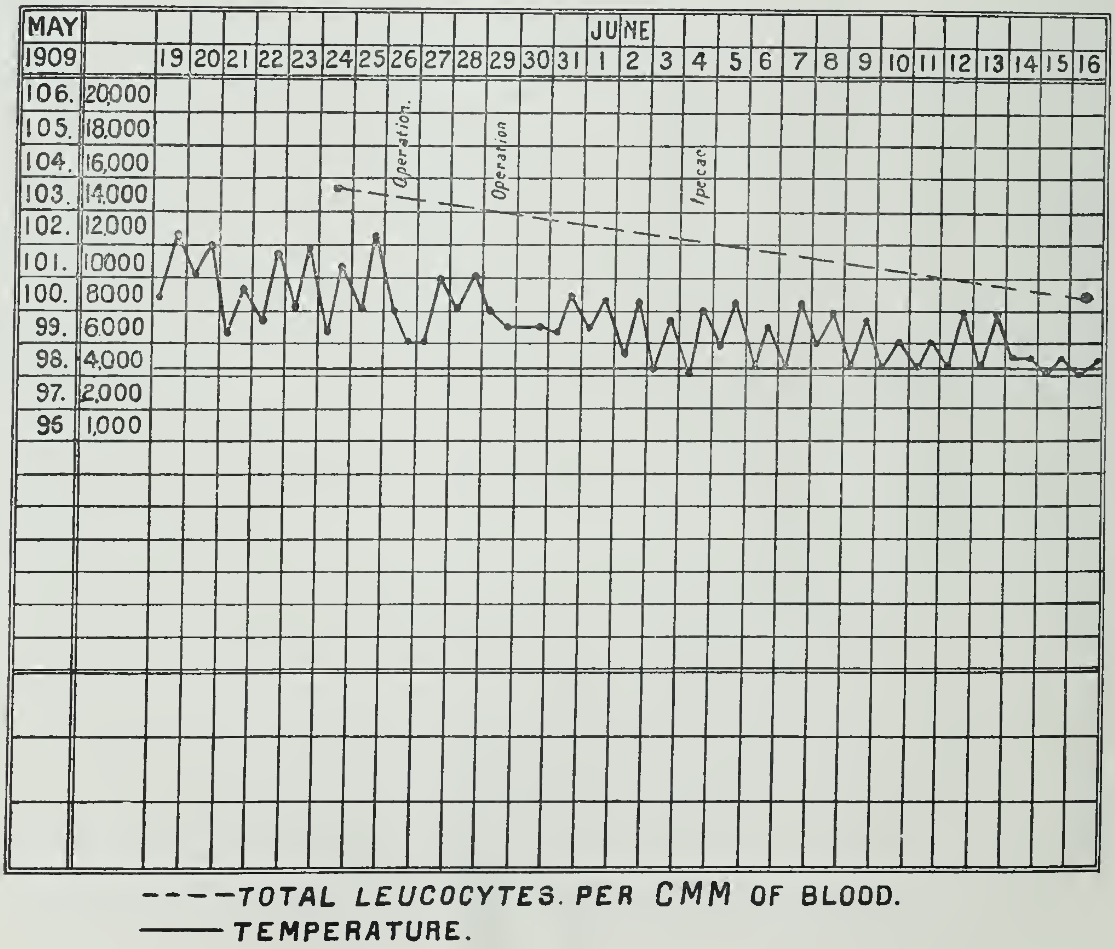
May 24th, 1909.—Admitted to hospital on 19th May complaining of pains across the right hypochondrium of two days' duration ; awoke suddenly in the night with pain, now constant.

Previous health good, no history of dysentery, had never seen mucus in his stools.

Social condition.—Arrived from Rangoon on 16th April.

May 26th, 1909.—Operation for liver abscess, pus removed. Profuse sweating at night.

CHART X.



May 29th, 1909.—Operation second abscess incised and drained.

June 22nd, 1909.—Patient is now doing well, wound healthy; temperature normal.

August 26th, 1909.—Discharged.

Microscopic examination of pus showed the presence of amœbæ and staphylococci. Examination of stools by culture showed the presence of amœbæ.

Treatment.—Operation, temperature remaining high, Pulv. ipecac was commenced on June 4th. Dose at first was gr. X, thrice daily, which was increased to gr. XX, twice daily, and this was finally increased to gr. XXX daily.

The chart on page 44 shows the course of the disease and the total number of W. B. C. per c.m.m. of blood :—

The following table shows the result of the examination of the blood :—

Name.	Age.	Date of examination.	W. B. C.	PERCENTAGES.				Parasites in blood—malaria.
				P. N.	S. M.	L. M.	E.	
T. . . .	..	24th May 1909 .	15,000	84	12	4	0	
		22nd June 1909 .	9,240	50	39	10	1	

REMARKS.—This case illustrates several points of very considerable practical importance in connection with the treatment of liver abscess. The patient had two abscesses opened one after the other, but in spite of this the temperature remained high. It is noteworthy that the administration of ipecacuanha in full doses reduced the temperature, and the leucocytic curve fell also. It is probable that this patient had an extensive amœbic invasion of his liver, as evidenced by the presence of two abscesses, and the action of the ipecacuanha was to limit the further amœbic extension. It is important, therefore, to bear in mind that ipecacuanha is not only of use before the formation of the liver abscess, but, also, after the abscess has been opened to prevent the further extension of the amœbic infection.

CASE ILLUSTRATING GROUP 2.

The following cases are given to illustrate the occurrence of a high leucocytosis in cases of amœbic dysentery :—

Case No. 208. F. C., European, age 20.

May 17th, 1909.—Admitted to hospital on 8th May complaining of diarrhœa with pains in the abdomen and straining, stools small, consisting of mucus and blood. Duration six days. The onset was gradual with ordinary looseness.

Previous health.—Good, no dysentery.

Present condition.—Patient well nourished and well developed. Tongue covered with a thick yellowish fur extending to tip and edges. Abdomen slightly retracted, and tenderness on pressure all along course of colon, most marked about sigmoid and cæcum ; neither *liver* nor *spleen* enlarged.

May 20th, 1909.—Condition improving. Motions 3-6 daily.

June 3rd, 1909.—Temperature rose on 1st June to 102·4, no rigors, 3-4 motions daily.

June 7th, 1909.—Again improved, one motion daily.

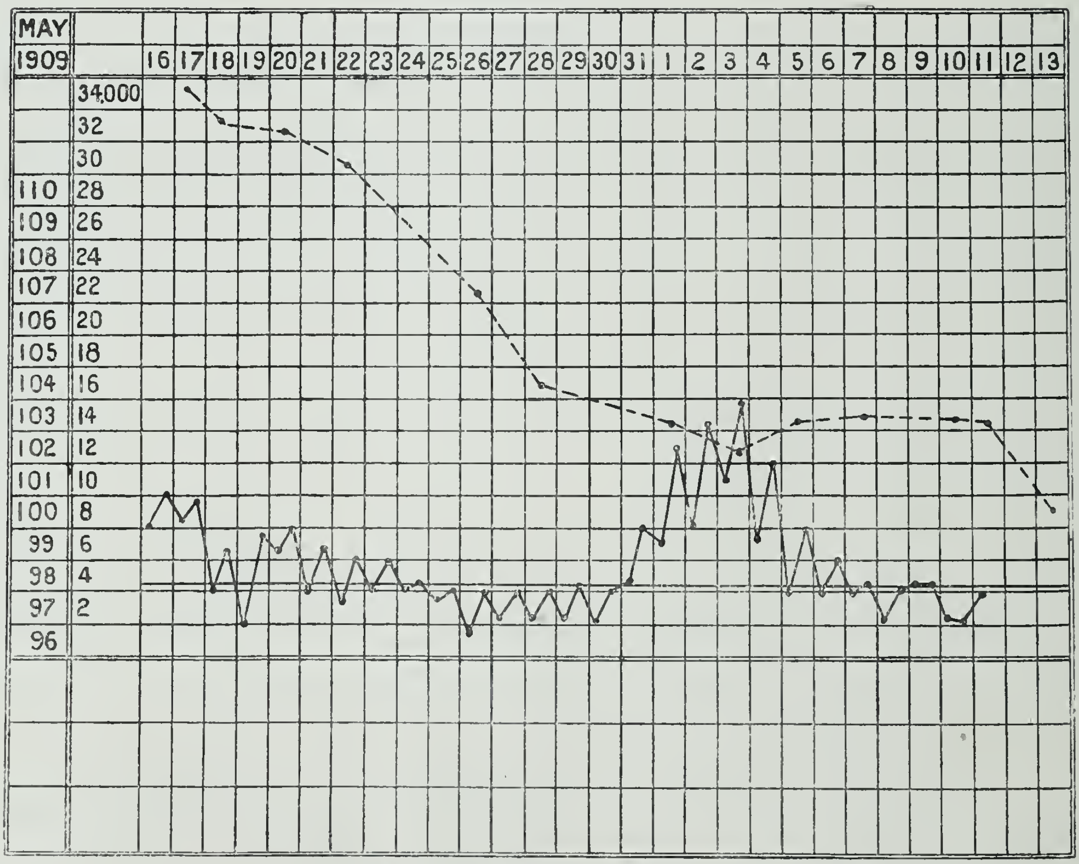
June 19th, 1909.—Temperature normal; tongue clean, one motion daily. He is able to walk about.

June 23rd, 1909.—Temperature normal. Has had slight griping pain occasionally; has three stools a day, with small amount of mucus and blood.

July 3rd, 1909.—Patient feels fairly well; no pain. One motion daily. Looks pale and debilitated. Sent to England on sick leave. Examination of the stools by culture showed the presence of amœbæ.

The following chart shows the course of the disease and the total number of W. B. C. per c.m.m. :—

CHART XI.



TOTAL LEUCOCYTES. PER CMM OF BLOOD.
TEMPERATURE.

The following table shows the result of the examination of the blood:—

Name.	Age.	Date of examination.	R. B. C.	W. B. C.	Percentages.				Parasites in blood—malaria.
					P. N.	S. M.	L. M.	E.	
C.	20	17th May 1909		35,240	86·5	10	2	1·5	—
		18th May 1909		33,850	81·5	12	5·5	1	
		20th May 1909		32,400					
		22nd May 1909		30,370					
		26th May 1909		22,130					
		28th May 1909		17,000					
		1st June 1909		14,860	80	16	3	1	
		3rd June 1909		13,480	79	16	5	0	
		5th June 1909		14,400					
		7th June 1909		15,920					
		9th June 1909		14,500					
		13th June 1909		10,000	57	37·5	3	2·5	
		17th June 1909			67	29	2·5	1·5	
		19th June 1909		15,640	51	43	5·5	0·5	
		21st June 1909		10,400	55	42	2	1	
		23rd June 1909		12,400	48·5	46·5	3	2	
		25th June 1909		12,000	49·5	45·5	3·5	1·5	
		27th June 1909		10,760	47	44	5·5	3·5	
		1st July 1909	3,625,000	9,640	47	45	5·5	2·5	

Remarks—This was a severe case of dysentery. The whole length of the colon was involved as shown by the tenderness on pressure. In this case the total leucocytes were greatly increased in number. Therefore when the lesion of the large intestine is extensive and severe a high degree of leucocytosis may be produced. It is necessary to bear this fact in mind in the differential diagnosis between a severe dysenteric lesion in the bowel and dysenteric complications outside the large intestine.

Case No. 216 G., Hindu, age 36.

May 21st, 1909.—Admitted on 20th May complaining of griping pains in the abdomen and frequent small motions, composed of mucus and blood. Duration ten days.

Previous health.—Good; no history of dysentery.

Social condition.—Has been in Bombay one year, formerly in Calcutta ; was in South Africa.

Present condition.—Well nourished, abdomen retracted. Tenderness on pressure all along the course of the colon. *Liver* and *spleen* not enlarged.

May 23rd, 1909.—Motions 5-6 in 24 hours. Pain in abdomen severe. Repeated vomiting. Tongue fairly clean.

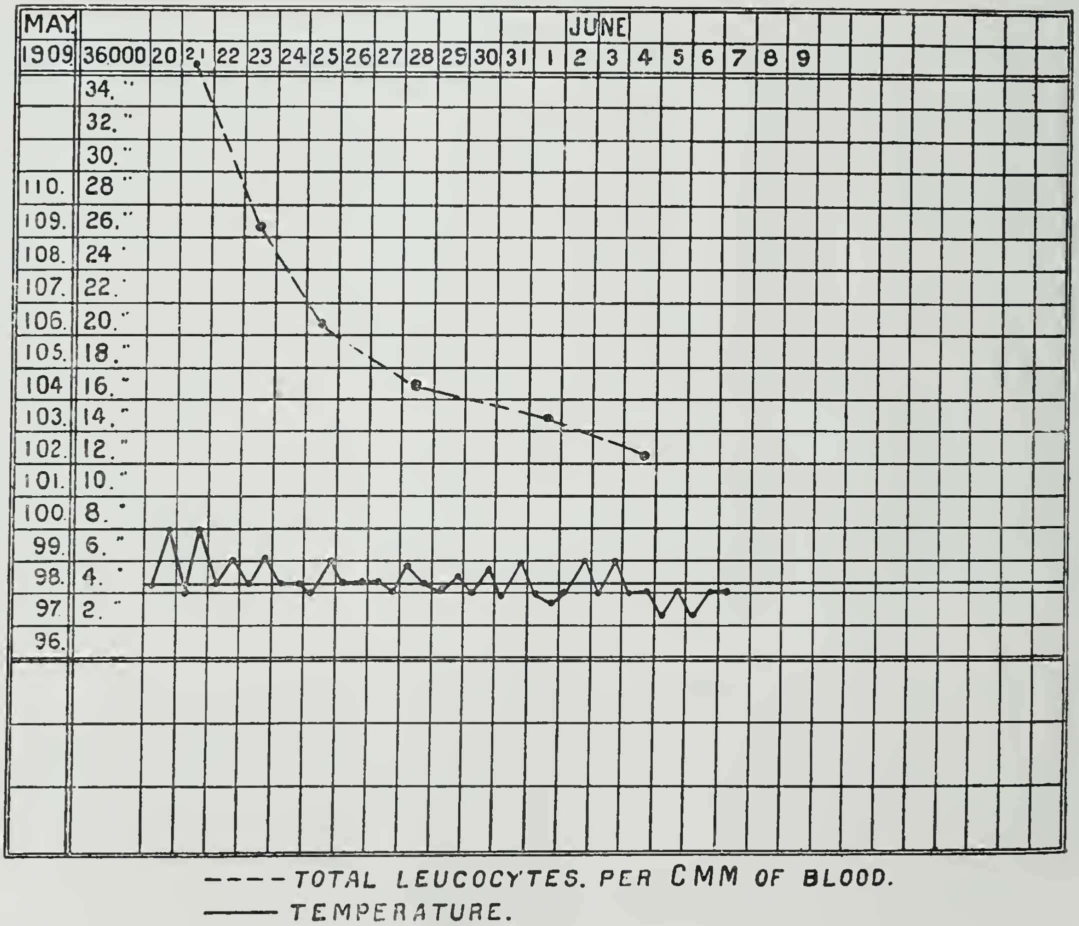
May 31st, 1909.—Tongue clean, no vomiting. Pain about umbilicus and right iliac fossa. Motions 5-6 daily.

June 4th, 1909.—Tongue clean, 3-5 loose motions daily.

June 6th, 1909.—Feels well, 1-2 motions daily. Discharged. Examination of his stools by microscope and by culture showed the presence of amœbæ.

The following chart shows the course of the disease and the total number of W. B. C. per c.m.m. of blood :—

CHART XII.



The following table shows the result of the examination of the blood :—

Name	Age.	Date of examination.	R. B. C.	W. B. C.	PERCENTAGES.				Parasite in blood—malaria.
					P. N.	S. M.	L. M.	E.	
G.	36	21st May 1909		36,720	75	20	3		—
		23rd May 1909	6,416,000	26,140					
		25th May 1909	6,920,000	20,700	61	34	3	2	
		28th May 1909		17,280	63·5	31	4·5	1	
		1st June 1909		15,200	59	37·5	3·5	0	
		4th June 1909		13,200					

REMARKS.—This case, also, had a severe and extensive lesion of the colon, and there was considerable pain. The leucocytosis was very high, but gradually fell as the symptoms improved. It may be that the severity of the infection in these cases is due to higher virulence of the particular strain of amœba. This opens up an interesting field for investigation, but, at the moment, there does not appear to be any method of conclusively deciding this point experimentally.

The general effects of amœbic infection have received hitherto little attention, and in the above study of the blood changes, etc., an attempt has been made to open up this important line of investigation. An interesting problem to determine is the relationship of hepatic disease, functional and organic, apart from hepatitis and liver abscess, to amœbic infection. It is known that cirrhotic changes in the liver are common in India. A careful study of the fauna of the intestine, and, particularly, of the amœbæ, might throw more light on this important subject, just as the investigation of the intestinal flora has led to many new ideas.

VI.—Diagnosis of Amœbic Infection.

The diagnosis can be made only by determining the presence of the amœba. This is by no means an easy matter, and it is necessary to be familiar with the appearance of the parasite at its different stages. A glance at the plates (Nos. I and II) will show the different appearances ; the size varies from that of a red blood corpuscle up to 30-40 microns in diameter. Further, the parasite at times is non-motile, and hence it is often difficult to distinguish it from other cells. Accordingly a considerable amount of experience is required before an accurate diagnosis in all cases can be given. It is always desirable to examine microscopically fresh material in the first place, subsequently the specimen may be stained if it is desired to study structural characters. The cultivation of amœbæ as a means of diagnosis must be regarded as still in its experimental stage. Cultivation affords a very simple and easy means of detecting amœbæ in the stools. The method adopted by us was as follows :—

The medium of Musgrave and Clegg was poured into small Petri plates and allowed to dry. A loopful of the stool was taken and parallel strokes were made on the surface of the agar with a platinum loop. The plates were set aside at room temperature (average 79°F.) They were daily examined under a low power of the microscope, and if present, the amœbæ were observed readily growing out from bacteria on the stroke. We made a parallel series of observations on the detection of amœbæ in the stools, namely, by microscopic examination and by cultivation, the object of the observations being to ascertain whether we could cultivate an amœba from a stool in which, after careful microscopic examination, we were unable to detect it, or it was present in a non-motile condition making its determination difficult.

The following table shows the results of the examination of the stools of a series of cases of dysentery :—

Serial No.	Number of case.	Date of admission to hospital.	Date of examination of stool.	Amœba.	
				Microscopic examination.	Cultivation.
1	167 C	29th March 1909	31st March 1909	+	+
2	66 M	27th March 1909	1st April 1909	+	+
3	171 C	31st March 1909	1st April 1909	+	+
4	176 C	4th April 1909	7th April 1909	+	+
5	71 M	31st March 1909	7th April 1909	+	+
6	183 C	12th April 1909	13th April 1909	—	+
7	73 M	13th April 1909	15th April 1909	—	+
8	186 C	12th April 1909	16th April 1909	—	+
9	74 M	12th April 1909	16th April 1909	+	+
10	75 M	15th April 1909	15th April 1909	+	+
11	77 M	12th April 1909	17th April 1909	—	+
12	185 C	10th April 1909	15th April 1909	—	+
13	78 M	16th April 1909	21st April 1909	—	+
14	191 C	23rd April 1909	26th April 1909	—	+
15	80 M	22nd April 1909	26th April 1909	—	+
16	89 M	29th April 1909	30th April 1909	—	+
17	201 C	2nd May 1909	3rd May 1909	—	+
18	203 C	3rd May 1909	4th May 1909	—	+
19	204 C	4th May 1909	5th May 1909	—	+
20	206 C	19th April 1909	8th May 1909	—	+
21	82 M	8th May 1909	10th May 1909	—	+
22	208 C	12th May 1909	14th May 1909	—	+
23	218 C	20th May 1909	22nd May 1909	—	+
24	213 C	18th May 1909	24th May 1909	—	+
25	216 C	20th May 1909	21st May 1909	+	+
26	220 C	28th May 1909	29th May 1909	—	+
27	222 C	2nd June 1909	3rd June 1909	—	+
28	84 C	30th May 1909	1st June 1909	—	+
29	224 C	6th June 1909	8th June 1909	—	+

This table shows that the amœba could be recovered from the stool in every case in which it had been determined microscopically and, also, in 21 cases out of the 29 in which it could not be determined by microscopic examination. Hence cultivation gives a more accurate estimate of the total amœbic content of the stools than microscopic examination. The relationship of cultivable amœbæ to the etiology of dysentery is a matter for further investigation, but the method of cultivation affords an excellent means for the study of amœbæ of the intestine.

This method of observation was applied, also, to the liver pus, and the following table shows the results of the observations on the pus from a series of cases of liver abscess:—

Serial No.	Number of case.	Date of operation.	Date of examination of pus.	Amœba.	
				Microscopic.	Cultivation.
1	196 C	24th April 1909 . .	29th April 1909 . .	+	+
2	193 C	25th April 1909 . .	1st May 1909 . .	+	+
3	217 C	20th May 1909 . .	29th May 1909 . .	—	+
4	219 C	22nd May 1909 . .	29th May 1909 . .	+	—

In order to determine whether amœbæ could be cultivated from the stools of patients suffering from liver abscess, but without symptoms of dysentery, the following table is given:—

Serial No.	Number of case.	Date of operation.	Date of examination of stools.	Amœba.	
				Microscopic examination.	Cultivation.
1	196 C	24th April 1909 . .	29th April 1909 . .	—	+
2	193 C	25th April 1909 . .	1st May 1909 . .	—	+
3	217 C	20th May 1909 . .	29th May 1909 . .	—	+
4	219 C	22nd May 1909 . .	29th May 1909 . .	—	+
5	85 M	26th May 1909 . .	26th May 1909 . .	—	+

It will be seen from this table that the amœba could be cultivated from the stools of patients suffering from liver abscess, but with no symptoms of dysentery, and the microscopic examination failed to show the presence of amœbæ.

The diagnosis of the early stage of amœbic invasion of the liver has been discussed already, and the importance of careful study of the blood changes, as a means of recognising these cases, should be remembered.

VII.—General Conclusions.

1. That bacillary dysentery occurs in Bombay, but the incidence, so far as our observations show, is low.

2. That dysentery associated with amœbæ is more prevalent.

3. That amœbæ can be cultivated from the stools of dysentery cases and the pus of liver abscess cases and grown outside the human body on a suitable medium.

4. That the prevalence of amœbæ shows a marked seasonal variation. The curve of amœbic prevalence closely follows the humidity curve and has no definite relationship to the temperature curve in Bombay.

5. That the season of greatest prevalence of amœbæ in Bombay corresponds to the season of greatest prevalence of dysentery as shown by the admission rate for dysentery for ten years in the Jails of Bombay.

6. That the amœbæ cultivated from the stools of dysentery cases and pus of iver abscess cases are not *Ent. histolytica* or *coli*, but are probably the same or similar forms to the amœbæ cultivated by Noc from dysentery and liver abscess cases in Cochin China.

7. That the same, or apparently the same, amœbæ can be cultivated from tap water.

8. That amœbæ can be cultivated from the stools of a large number of normal animals.

9. That the amœba forms cysts both inside and outside the body to resist conditions unfavourable to its life, and that the cystic stage is important both in regard to the treatment and prevention of the disease, and cases of dysentery should not be considered cured until the stools are found to be free from cysts.

10. That amœbæ may be cultivated from the fæces of a person suffering from abscess of the liver, although he at the time shows no signs of dysentery.

11. That the evidence at present available indicates that water is a channel by which amœbæ of dysentery may be disseminated in endemic areas.

12. That a high leucocytosis, in cases of amœbic dysentery with an extensive lesion of the colon, may occur.

13. That a moderate leucocytosis, with little alteration of the relative percentages, may occur in cases of slight amœbic dysentery, or in cases where, at the time of examination, no symptoms of dysentery exist. This associated with rise of temperature may indicate an early hepatic invasion by amœbæ; other signs of

hepatitis may or may not be present at first, but sooner or later will develop, and, if not treated, may culminate in the grave complication of abscess of the liver.

14. That the administration of ipecacuanha in full doses, as pointed out by Major L. Rogers, I.M.S., in the class of case referred to in 13 will produce a reduction to normal of the temperature and leucocyte curves and prevent the formation of liver abscess. These observations are of particular importance in regard to the treatment of dysentery among the European troops in India, amongst whom amœbic abscess of the liver is, next to enteric fever, the most fatal disease which has to be dealt with. (In 1908 enteric fever caused 188 deaths and abscess of the liver 55 deaths in the European army in India.)

15. The specific identity of the amœbæ which are demonstrable microscopically in the fresh stools with those appearing in cultures from the same material does not seem to be clearly established, and the interpretation of the results of cultivation of amœbæ from the fæces presents peculiar difficulties.

Doflein[1], after considering the various amœboid organisms which have been described as occurring in the fæces, dwells upon the difficulty of coming to a decision as to which of the following headings such organisms should come under :—

(*i*) "*Gelegentliche Passanten des Darms*", i.e., casual visitors in the gut, or pseudo-parasites. This group would include free-living amœbæ swallowed in the encysted condition and excreted unchanged, as Werner[2] described for *Amœba limax* in the case of the house-fly;

(*ii*) Facultative parasites; and

(*iii*) Amœbæ specifically adapted to the parasitic habit.

Moreover, from our observations of the occurrence of amœbæ in cultures from tap water (p. 19), and from the excreta of flies (p. 21), it seems that specimens of fæces collected in the ordinary way are open to contamination with amœbæ *outside the body*, e.g., from the walls of the containing vessel and from direct deposit by flies.

Since the report went to press, one of us (R. T. W.) has found that growths of amœboid organisms may appear in association with moulds and other bacteria collected by exposing plates of uninseminated Musgrave's agar to the air.

These observations, which might indicate a further fallacy in cultivation experiments, applicable not only to the fæces but to liver abscess, pus, and to any other material, will be included in a later report.

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[1] Doflein.—Lehrbuch der Protozœnkunde 1909, p. 514.

[2] Werner.—Beihefte Z. Archiv f. Schiff u. Trophyg. Vol. XII, No. 11, 1908

PART II.

APPENDIX.

Table I.

Showing results obtained by observations on cases of Bacillary Dysentery.

NOTE.—In this table are recorded the daily observations on a number of cases of bacillary dysentery. The fæces of each case were examined for a considerable period after the stools had become feculent. (*Vide* page 9 of this Report for a discussion of results.)

BACILLARY DYSENTERY, CASE NO. 1.

Rank.—No. 976, Musician.

Name.—R. R.

Regiment.—116th Mahrattas.

Date of observation.	Character of stool.	DYSENTERY BACILLI IN STOOL.	
		Shiga.	Flexner.
22nd January 1909	Blood and mucus		+
23rd January 1909	Ditto		—
25th January 1909	Ditto		+
26th January 1909	Ditto		+
27th January 1909	Ditto		+
28th January 1909	Ditto		+
30th January 1909	Partly feculent		+
3rd February 1909	Ditto		—
4th February 1909	Feculent semi-solid . . .		—
5th February 1909	Ditto . . .		—
6th February 1909	Feculent		—
8th February 1909	Ditto		—
9th February 1909	Ditto		—
10th February 1909	Ditto		—
11th February 1909	Ditto		—
12th February 1909	Ditto		—
13th February 1909	Ditto		—
16th February 1909	Ditto		—

REMARKS.—This was a mild case of dysentery. He had suffered from simple diarrhœa for three days before admission to hospital, on 26th January 1909. He was relieved in about three days by treatment with Magnesium sulphate. He has been in Bombay for the past year. This demonstrates the result of an infection with B. dysenteriæ (Flexner).

BACILLARY DYSENTERY, CASE NO. 2.

Rank.—No. 27336, Gunner.

Name.—M.

Regiment.—R. A.

Date of observation.	Character of stool.	DYSENTERY BACILLI IN STOOL.	
		Shiga.	Flexner.
9th January 1909	Blood and mucus	—	
11th January 1909	Ditto	—	
16th January 1909	Ditto	+	
19th January 1909	Ditto	—	
22nd January 1909	Ditto	+	
23rd January 1909	Ditto	—	
25th January 1909	Ditto	—	
28th January 1909	Ditto	—	
29th January 1909	Ditto	+	
30th January 1909	Ditto	+	
1st February 1909	Ditto	—	
3rd February 1909	Ditto	—	
4th February 1909	Ditto	—	
5th February 1909	Mucus shreds	—	
6th February 1909	Mucus	—	
8th February 1909	Ditto	—	
9th February 1909	Ditto	—	
10th February 1909	Ditto	—	
12th February 1909	Ditto	—	
15th February 1909	Ditto	—	
16th February 1909	Feculent	—	
26th February 1909	Ditto	—	
27th February 1909	Ditto	—	
1st March 1909	Ditto	—	

REMARKS.—This patient had dysentery in October last at Kirkee. The present illness is a relapse. He has an intermittent temperature. This case illustrates the results of a Shiga infection. The signs and symptoms were more severe and the illness more proonged than in the case of an infection with Flexner's bacillus.

BACILLARY DYSENTERY, CASE NO. 3.

Rank.—No. 2053, Sepoy.

Name.—F. M.

Regiment.—109th Infantry.

Date of observation.	Character of stool.	DYSENTERY BACILLI IN STOOL.	
		Shiga.	Flexner.
7th December 1908	Blood and mucus	+	
9th December 1908	Ditto	—	
10th December 1908	Mucus	+	
11th December 1908	Feculent	—	
12th December 1908	Ditto	—	
14th December 1908	Ditto	—	

REMARKS.—This case is one of infection by B. dysenteriæ (Shiga). It was a first attack and was treated early with the result that the symptoms rapidly cleared up.

BACILLARY DYSENTERY, CASE NO. 4.

Rank.—

Name.—V. R.

Regiment.—Police Constable, Bombay.

Date of observation.	Character of stool.	DYSENTERY BACILLI IN STOOL.	
		Shiga.	Flexner.
4th December 1909	....	+	

REMARKS.—This was a very mild case of Shiga infection and readily responded to treatment.

BACILLARY DYSENTERY, CASE NO. 5.

Rank.—
Name.—K. S.
Regiment.—Police Constable, Bombay.

Date of observation.	Character of stool.	DYSENTERY BACILLI IN STOOL.	
		Shiga.	Flexner.
4th December 1908	....	+	

REMARKS.—This case was also of a mild type. It is probable that such cases, presenting few symptoms and causing the patient little or no inconvenience, are the means of maintaining the existence of the bacillus. Many such cases must never come for treatment, and are in a position to infect others.

BACILLARY DYSENTERY, CASE NO. 6.

Rank.—
Name.—S. S.
Regiment.—Police Constable, Bombay.

Date of observation.	Character of stool.	DYSENTERY BACILLI IN STOOL.	
		Shiga.	Flexner.
14th January 1909	Blood and mucus	..	+
20th January 1909	Ditto	..	+
21st January 1909	Ditto	..	+
22nd January 1909	Ditto	..	+
25th January 1909	Ditto	..	+
26th January 1909	Ditto	..	+
27th January 1909	Ditto	..	—
28th January 1909	Semi-solid feculent	..	—
29th January 1909	Ditto	..	—
30th January 1909	Ditto	..	+
10th February 1909	Feculent	..	—
11th February 1909	Ditto	..	—
16th February 1909	Ditto	..	—
17th February 1909	Ditto	..	—
19th February 1909	Ditto	..	—

REMARKS.—It will be noted that in this case the excretion of the bacilli ceased when the stools became feculent. No recurrence was noted.

BACILLARY DYSENTERY, CASE NO. 7.

Rank.—Sepoy, No. 1806.

Name.—A. K.

Regiment.—109th Infantry.

Date of observation.	Character of stool.	DYSENTERY BACILLI IN STOOL.	
		Shiga.	Flexner.
27th February 1909	Blood and mucus.	..	+
1st March 1909	Ditto	..	+
2nd March 1909	Ditto	..	+
3rd March 1909	Ditto	..	+
4th March 1909	Ditto	..	+
5th March 1909	Feculent	..	—
11th March 1909	Ditto	..	—
12th March 1909	Feculent (formed)	..	—
13th March 1909	Ditto	..	—
15th March 1909	Ditto	..	
22nd March 1909	Ditto	..	—
23rd March 1909	Ditto	..	—
24th March 1909	Ditto	..	—
25th March 1909	Ditto	..	—
26th March 1909	Ditto	..	—
29th March 1909	Ditto	..	—
30th March 1909	Ditto	..	—
31st March 1909	Ditto	..	—
1st April 1909	Ditto	..	—
2nd April 1909	Ditto	..	—
3rd April 1909	Ditto	..	—

REMARKS.—This case illustrates the type of dysentery frequently met with amongst the Native troops. Under treatment the symptoms quickly cleared up and the stools became normal. It will be noted that the bacilli were not found after the stools became formed. This man was ill in the lines for three days before admission. This is a common history in cases of dysentery and has an important bearing on the prevention of the disease. This man had no fever during his illness.

BACILLARY DYSENTERY, CASE No. 8.

Rank.—No. 933, Sepoy.
Name.—H. J.
Regiment.—116th Mahrattas.

Date of observation.	Character of stool.	DYSENTERY BACILLI IN STOOL.	
		Shiga.	Flexner.
4th January 1909	Blood and mucus	..	—
6th January 1909	Ditto	..	—
7th January 1909	Ditto	..	—
19th January 1909	Ditto	..	—
20th January 1909	Ditto	..	—
21st January 1909	Ditto	..	—
22nd January 1909	Ditto	..	—
23rd January 1909	Ditto	..	—
25th January 1909	Ditto	..	—
26th January 1909	Ditto	..	—
27th January 1909	Ditto	..	—
28th January 1909	Ditto	..	—
29th January 1909	Ditto	..	—
30th January 1909	Ditto	..	—
1st February 1909	Ditto	..	+
3rd February 1909	Ditto	..	—
4th February 1909	Ditto	..	—
5th February 1909	Ditto	+ Amœbæ	—
6th February 1909	Ditto	..	—
8th February 1909	Ditto	..	—
9th February 1909	Feculent	..	—
10th February 1909	Ditto	..	—
11th February 1909	Ditto	..	—
12th February 1909	Ditto	..	—

REMARKS.—This is an instructive case. The patient, whilst on field training, developed dysentery and was sent to hospital. The symptoms were sub-acute. At first he was given Mag. sulphate without benefit; on the 6th February 1909 he was placed on ipecacuanha and he rapidly recovered. The bacteriological findings were instructive; for some time no dysentery bacilli were cultivated, but on one occasion, 1st February 1909, Flexner's bacillus was recovered; on the 5th February 1909 amœbæ were detected in the stools by microscopic examination. The fact that the symptoms cleared up under ipecacuanha points to their being due to amœbic rather than bacillary infection.

BACILLARY DYSENTERY, CASE NO. 9.

Rank.—Sepoy No. 2287.

Name.—J. K.

Regiment.—109th Infantry.

Date of observation.	Character of stool.	DYSENTERY BACILLI IN STOOLS.	
		Shiga.	Flexner.
7th January 1909	Mucus and blood . . .	..	+
8th January 1909	Ditto	..	—
11th January 1909	Ditto	..	—
12th January 1909	Ditto	..	+
15th January 1909	Ditto	..	+
16th January 1909	Ditto	..	—
18th January 1909	Ditto	..	—
19th January 1909	Ditto	..	—
23rd January 1909	Ditto	..	+
25th January 1909	Ditto	..	+
26th January 1909	Ditto	..	+
27th January 1909	Ditto	..	+
28th January 1909	Feculent	..	—
30th January 1909	Ditto	..	—
1st February 1909	Feculent, some mucus . .	..	—
3rd February 1909	Ditto	..	—
4th February 1909	Ditto	..	—
5th February 1909	Feculent	..	—
6th February 1909	Ditto	..	—
8th February 1909	Ditto	..	—
9th February 1909	Ditto	..	—
10th February 1909	Ditto	..	—
12th February 1909	Ditto	..	—
16th February 1909	Ditto	..	—

REMARKS.—The symptoms were sub-acute with a small quantity of blood in the stools. The temperature was normal throughout. His condition improved under treatment with Mag. sulphate. It is noteworthy that no bacilli were detected in the stools after they became feculent.

BACILLARY DYSENTERY, CASE NO. 10.

Rank.—Sepoy No. 1032.
Name.—A. G.
Regiment.—116th Mahrattas.

Date of observation.	Character of stool.	DYSENTERY BACILLI IN STOOLS.	
		Shiga.	Flexner.
15th January 1909	Blood and mucus	..	—
18th January 1909	Ditto	..	+
21st January 1909	Ditto	..	—
22nd January 1909	Ditto	..	+
23rd January 1909	Ditto	..	—
25th January 1909	Ditto	..	—
26th January 1909	Ditto	..	+
27th January 1909	Ditto	..	+
28th January 1909	Ditto	..	—
29th January 1909	Feculent	..	—
30th January 1909	Ditto	..	—
1st February 1909	Feculent, mucus present	..	+
3rd February 1909	Ditto	..	—
4th February 1909	Feculent	..	—
5th February 1909	Ditto	..	—
6th February 1909	Ditto	..	—
8th February 1909	Ditto	..	—
10th February 1909	Ditto	..	—
11th February 1909	Ditto	..	—
12th February 1909	Ditto	..	—
13th February 1909	Ditto	..	—
15th February 1909	Ditto	..	—
16th February 1909	Ditto	..	—
17th February 1909	Ditto	..	—

REMARKS.—This patient developed slight symptoms of dysentery whilst under treatment for gonorrhœa on 21st November 1908. Under Mag. sulphate the symptoms of dysentery improved. He had a relapse on 14th January 1909. The bacilli in this case disappeared from the stools on their becoming feculent.

BACILLARY DYSENTERY, CASE No. 11.

Name.—R. F.

Hospital.—St. George's.

Date of observation.	Character of stool.	DYSENTERY BACILLI IN STOOL.	
		Shiga.	Flexner.
23rd January 1909	Trace of blood and mucus . .	..	+
25th January 1909	Mucus	..	—
26th January 1909	Mucus (feculent)	..	—
27th January 1909	Ditto	..	—
28th January 1909	Ditto	..	—
29th January 1909	Ditto	..	—
30th January 1909	Ditto	..	+
4th February 1909	Ditto	..	—
5th February 1909	Ditto	..	—
6th February 1909	Ditto	..	—
8th February 1909	Feculent	..	—
9th February 1909	Ditto	..	—

REMARKS.—This patient had suffered from attacks of "diarrhœa," which lasted two—three days, for the last two years. On admission to hospital he was suffering from fever and diarrhœa. He had benign tertian parasites in his blood. Flexner's bacillus was found on several occasions in his stools: the ova of ankylostoma were present also.

BACILLARY DYSENTERY, CASE No. 12.

Name.—M. A.

Hospital.—J. J. Hospital.

Date of observation.	Character of stool.	DYSENTERY BACILLI IN STOOL.	
		Shiga.	Flexner.
25th January 1909	Mucus and blood	..	+
26th January 1909	Ditto	..	+
27th January 1909	Ditto	..	+
28th January 1909	Ditto	..	+
29th January 1909	Feculent, semi-fluid . . .	..	+
1st February 1909	Ditto	..	—

REMARKS.—This patient left hospital on the 1st February 1909. There was nothing noteworthy in his case.

BACILLARY DYSENTERY, CASE NO. 13.

Name.—K.

Hospital.—St. George's.

Date of observation.	Character of stool.	Dysentery bacilli in stool.	
		Shiga.	Flexner.
28th January 1909	Watery feculent (Pulv. jalap given)	+	
29th January 1909	Watery feculent	+	
30th January 1909	Ditto	—	
1st February 1909	Fluid	—	
3rd February 1909	Ditto	—	
4th February 1909	Ditto	—	
5th February 1909	Ditto	—	
8th February 1909	Ditto	—	
9th February 1909	Ditto	—	
10th February 1909	Ditto	—	
11th February 1909	Feculent	—	
9th March 1909	Ditto	—	

REMARKS.—This case is interesting. The patient is a Japanese. He was not suffering from dysentery at the date of examination. His urine contained a large quantity of albumen; he had œdema of the face. He had no rise of temperature on his admission. He was given purgatives, Pulv. jalap co. His stools were examined and the Shiga bacillus recovered on two occasions. This observation is important from the point of view of the prevention of the disease, as the patient had no symptoms of dysentery acute or chronic.

Table II.

Showing various sources at Bombay from which Amœbæ were cultivated.

NOTE.—In this table are recorded the results of the examination of material for amœbæ. The material was varied and obtained from different sources. It will be observed that the distribution of the amœba, apart from dysentery and liver abscess, is wide. It was recovered from saliva, from pus removed from a hip joint abscess, and a lumbar abscess. (*Vide* page 13 of this Report for a discussion of the results.)

Serial No.	Nature of material.	Nature of case from which material obtained.	No. of cases examined.	Amœbæ in
1	Sputum	Tubercle	2	2
2	Sputum	Liver abscess	1	1
3	Sputum	Dysentery	2	2
4	Saliva	Healthy	8	4
5	Pus (not sterile)	Hip-joint abscess	1	1
6	Pus (not sterile)	Lumbar abscess	1	1
7	Pus (not sterile)	Empyæma	1	0
8	Water (drinking)	Bombay tap water	4	4
9	Stools	Healthy	2	2
10	Stools	Diarrhœa	1	0
11	Stools	Malaria	1	1
12	Stools	Scabies	1	0
13	Stools	Fever (ordinary)	1	1
14	Stools	Piles	1	1
15	Stools	Wound, simple	1	1

Table III.

Showing the result of the examination of the stools of a number of healthy monkeys for Amœbæ.

NORMAL MONKEY.

NOTE.—In this table are recorded the results of the examination of normal monkeys for the presence or absence of amœbæ in their stools, and the record of the body weight of the animals throughout the observations. As monkeys have been employed for feeding experiments with amœbæ it was extremely important to determine whether or not amœbæ were present normally in their stools. (*Vide* pages 27 and 30 of this Report for a discussion of the results.)

Number of monkey.	Weight (grams).	Stools.	Amœbæ.	
			Microscope.	Cultivation.
10	1,800—17th May 1909		— 17th May 1909 Trichocephalus ova.	— 17th May 1909.
	1,850—19th May 1909		— 18th May 1909 Trichomonas.	— 20th May 1909.
	1,900—22nd May 1909		— 19th May 1909	— 22nd May 1909.
	1,850—29th May 1909		— 22nd May 1909	— 24th May 1909.
	1,810—31st May 1909	No diarrhœa	— 24th May 1909 Trichocephalus ova.	— 15th June 1909.
	1,850—7th June 1909	Ditto	— 15th June 1909	+ 21st June 1909.
	1,810—14th June 1909	Ditto	— 21st June 1909	— 30th June 1909.
	1,790—21st June 1909	Ditto	— 30th June 1909	+ 6th July 1909.
	1,750—28th June 1909	Ditto	— 6th July 1909	..
	1,680—5th July 1909	Ditto	..	..
	1,610—12th July 1909	Ditto	..	..
	1,610—19th July 1909	Ditto	..	..
11	1,520—17th May 1909		— 17th May 1909	— 17th May 1909.
	1,550—19th May 1909		— 19th May 1909	— 20th May 1909.
	1,580—22nd May 1909		— 22nd May 1909	— 22nd May 1909.
	1,550—24th May 1909		— 24th May 1909 Trichomonas.	— 24th May 1909.
	1,520—31st May 1909	No diarrhœa	— 15th June 1909	— 15th June 1909
	1,500—7th June 1909	Ditto	— 22nd June 1909 Trichomonas.	— 22nd June 1909.
	1,500—14th June 1909	Ditto	— 29th June 1909	— 29th June 1909.
	1,440—21st June 1909	Ditto	— 7th July 1909	+ 7th July 1909.
	1,500—28th June 1909	Ditto	— 14th July 1909	+ 14th July 1909.

Table III—*continued.*

NORMAL MONKEY.

Number of monkey.	Weight (grams).	Stools.	AMŒBÆ.	
			Microscope.	Cultivation.
	1,500—5th July 1909	No. diarrhœa.		
	1,470—12th July 1909	Ditto.		
	1,490—19th July 1909	Ditto.		
12	1,700—17th May 1909		— 17th May 1909 Trichomonas.	— 17th May 1909.
	1,750—19th May 1909		— 18th May 1909 Trichomonas.	— 20th May 1909.
	1,740—22nd May 1909		— 19th May 1909	— 22nd May 1909.
	1,750—24th May 1909		— 22nd May 1909	— 24th May 1909.
	1,690—31st May 1909	No diarrhœa	— 24th May 1909. Trichomonas.	+ 15th June 1909.
	1,760—7th May 1909	Ditto	— 15th May 1909	— 22nd June 1909.
	1,720—14th June 1909	Ditto	— 22nd June 1909	— 29th June 1909.
	1,770—21st June 1909	Ditto	— 29th June 1909	— 7th July 1909.
	1,800—28th June 1909	Ditto	— 7th July 1909	+ 14th July 1909.
	1,830—5th July 1909	Ditto	— 14th July 1909.	
	1,830—12th July 1909	Ditto.		
	1,850—19th July 1909	Ditto.		
14	1,420—26th May 1909		— 26th May 1909	— 26th May 1909.
	1,370—31st May 1909	Slight diarrhœa	— 16th June 1909	— 16th June 1909.
	1,450—7th June 1909	No diarrhœa	— 23rd June 1909	— 23rd June 1909.
	1,480—14th June 1909	Ditto	— 30th June 1909	+ 30th June 1909.
	1,470—21st June 1909	Ditto	— 9th July 1909	— 9th July 1909.
	1,470—28th June 1909	Ditto	— 15th July 1909 **Trichomonas.**	+ 15th July 1909.
	1,490—5th July 1909	Ditto.		
	1,460—17th July 1909	Ditto.		
	1,450—19th July 1909	Ditto.		
15	1,250—21st June 1909		— 23rd June 1909	— 23rd June 1909.

Table III—*continued.*

NORMAL MONKEY.

Number of monkey.	Weight (grams).	Stools.	AMŒBÆ.	
			Microscope.	Cultivation.
	1,140—28th June 1909	No diarrhœa	— 30th June 1909	+ 30th June 1909.
	1,170—5th July 1909	Ditto	— 9th July 1909	— 9th July 1909.
	1,160—12th July 1909	Ditto	— 15th July 1909 Trichomonas.	+ 15th July 1909.
	1,140—19th July 1909	Ditto		
16	1,260—21st June 1909		— 23rd June 1909	+ 23rd June 1909.
	1,120—28th June 1909	No diarrhœa	— 1st July 1909 Trichomonas.	— 1st July 1909.
	1,170—5th July 1909	Ditto	— 9th July 1909	— 9th July 1909.
	1,160—12th July 1909	Ditto	— 15th July 1909	+ 15th July 1909.
	1,160—19th July 1909	Ditto.		
17	2,030—28th June 1909		— 1st July 1909	— 1st July 1909.
	1,990—5th July 1909	No diarrhœa	— 10th July 1909	— 10th July 1909.
	1,990—12th July 1909	Ditto	— 12th July 1909 Trichomonas.	+ 12th July 1909.
	1,990—19th July 1909	Ditto.		
18	2,200—28th June 1909		— 1st July 1909	— 1st July 1909.
	1,830—5th July 1909	No diarrhœa	— 10th July 1909	— 10th July 1909.
	1,800—12th July 1909	Ditto	— 12th July 1909 Trichomonas.	— 12th July 1909.
	1,800—19th July 1909	Ditto	— 16th July 1909 Trichomonas.	+ 16th July 1909.
			— 19th July 1909 Trichomonas.	+ 20th July 1909.
19	1,370—5th July 1909.		— 10th July 1909	— 10th July 1909.
	1,280—12th July 1909	No diarrhœa	— 16th July 1909 Trichomonas.	+ 17th July 1909.
	1,310—19th July 1909	Ditto.		
20	1,770—5th July 1909		— 10th July 1909	— 10th July 1909.
	1,760—12th July 1909	No diarrhœa	— 16th July 1909 Trichomonas.	+ 17th July 1909.
	1,690—19th July 1909	Ditto.		
21	1,920—20th July 1909		— 22nd July 1909	+ 22nd July 1909.

Table III—*continued.*

NORMAL MONKEY.

Number of monkey	Weight (grams).	Stools.	AMŒBÆ.	
			Microscope.	Cultivation.
	1,810—26th July 1909	No diarrhœa	— 29th July 1909 Trichomonas.	+ 29th July 1909.
	1,640—2nd August 1909	Ditto	— 3rd August 1909 (Strongyloides).	3rd August 1909.
	1,570—9th August 1909	Diarrhœa, no blood	— 9th August 1909	— 9th August 1909.
22	2,320—20th July 1909		— 22nd July 1909	+ 22nd July 1909
	2,210—26th July 1909	No diarrhœa	— 29th July 1909 Trichomonas.	+ 29th July 1909.
	2,110—2nd August 1909	Ditto	— 3rd August 1909 Trichomonas.	— 3rd August 1909.
	2,130—9th August 1909	Ditto.		
23	1,710—23rd July 1909		— 23rd July 1909 Trichomonas.	+ 23rd July 1909.
	1,660—26th July 1909	No diarrhœa	— 30th July 1909	+ 30th July 1909.
	1,730—2nd August 1909	Ditto	— 4th August 1909	— 4th August 1909.
	1,760—9th August 1909	Ditto.		
24	1,530—23rd July 1909	Ditto	— 23rd July 1909 Strongyloides.	+ 23rd July 1909.
	1,540—26th July 1909	Ditto	— 30th July 1909 Trichomonas.	— 30th July 1909.
	1,510—2nd August 1909	Ditto	— 4th August 1909	— 4th August 1909.
	1,500—9th August 1909	Ditto.		
25	1,780—23rd July 1909	Ditto	— 23rd July 1909 Trichomonas.	+ 23rd July 1909.
	1,810—26th July 1909	Ditto	— 30th July 1909	+ 30th July 1909.
	1,770—2nd August 1909	Ditto	— 4th August 1909	— 4th August 1909.
	1,790—9th August 1909	Ditto.		
26	2,000—23rd July 1909	Ditto	— 24th July 1909	+ 24th July 1909.
	2,060—26th July 1909	Ditto	— 30th July 1909 Trichomonas.	— 30th July 1909.
	1,980—2nd August 1909	Ditto	— 4th August 1909	— 4th August 1909.
	1,990—9th August 1909	Ditto.		

Table III—*continued.*

NORMAL MONKEY.

Number of monkey.	Weight (grams).	Stools.	Amœbæ.	
			Microscope.	Cultivation.
27	1,270—23rd July 1909	No diarrhœa	— 26th July 1909.	+ 26th July 1909.
	1,300—26th July 1909	Ditto	— 31st July 1909	+ 31st July 1909.
	1,360—2nd August 1909	Ditto	— 5th August 1909 Trichomonas.	+ 5th August 1909.
	1,350—9th August 1909	Ditto.		
28	1,660—23rd July 1909	Ditto	— 26th July 1909	+ 26th July 1909.
	1,650—26th July 1909	Ditto	— 31st July 1909	+ 31st July 1909.
	1,650—2nd August 1909	Ditto	— 5th August 1909 Trichomonas.	— 5th August 1909.
	1,680—9th August 1909	Ditto.		
29	1,120—23rd July 1909	Ditto	— 26th July 1909.	+ 26th July 1909.
	1,170—26th July 1909	Ditto	— 31st July 1909	+ 31st July 1909.
	1,120—2nd August 1909	Ditto	— 5th August 1909 Trichomonas.	— 5th August 1909.
	1,179—9th August 1909	Ditto.		
30	1,990—23rd July 1909	Ditto	— 26th July 1909	+ 26th July 1909.
	1,980—26th July 1909	Ditto	— 31st July 1909. Trichomonas.	+ 31st July 1909.
	1,960—2nd August 1909	Ditto.	— 5th August 1909 Trichomonas.	+ 5th August 1909.
	1,990—9th August 1909	Ditto.		
31	3,130—5th February 1909.	Ditto	— 7th August 1909 Trichomonas.	+ 7th August 1909.
32	1,990—5th August 1909	Ditto	— 7th August 1909	— 7th August 1909.
	1,850—9th August 1909	Ditto	— 13th August 1909	+ 13th August 1909.
	1,830—16th August 1909	Ditto	— 20th August 1909	+ 20th August 1909.
	1,870—23rd August 1909.	Ditto	— 23rd August 1909	+ 23rd August 1909.
33	1,490—5th August 1909	Ditto	— 7th August 1909	+ 7th August 1909.
34	2,030—5th August 1909	Ditto	— 7th August 1909	— 7th August 1909.
	2,030—9th August 1909	Ditto	— 13th August 1909	+ 13th August 1909.
	1,920—16th August 1909	Ditto	— 20th August 1909	+ 20th August 1909.

Table III—*continued.*

NORMAL MONKEY.

Number of monkey.	Weight (grams).	Stools.	AMŒBÆ.	
			Microscope.	Cultivation.
	1,970—23rd August 1909.	No diarrhœa	— 24th August 1909 Trichomonas.	+ 24th August 1909.
35	1,910—9th August 1909	Ditto	— 14th August 1909	+ 14th August 1909.
	1,780—16th August 1909	Ditto	20th August 1909	+ 20th August 1909.
	1,790—23rd August 1909.	Ditto	— 25th August 1909 Trichomonas.	— 25th August 1909.
36	2,080—9th August 1909	Ditto	— 13th August 1909	+ 13th August 1909.
	1,990—16th August 1909.	Ditto	— 20th August 1909	+ 20th August 1909.
	2,020—23rd August 1909.	Ditto	— 25th August 1909	+ 25th August 1909.
37	1,870—13th August 1909.	Ditto	— 14th August 1909	+ 14th August 1909.
	1,750—16th August 1909.	Ditto	— 20th August 1909	+ 20th August 1909.
	1,840—23rd August 1909.	Ditto	— 25th August 1909	— 25th August 1909.
38	1,910—13th August 1909.	Ditto	— 14th August 1909 Trichomonas.	— 14th August 1909.
	1,820—16th August 1909.	Ditto	— 19th August 1909	+ 19th August 1909.
	1,800—23rd August 1909.	Ditto	— 21st August 1909 Trichomonas.	+ 21st August 1909.
			— 26th August 1909	+ 26th August 1909.
39	1,820—13th August 1909.	Ditto	— 14th August 1909 Trichomonas.	+ 14th August 1909.
	1,630—16th August 1909.	Ditto	— 21st August 1909	+ 21st August 1909.
	1,680—23rd August 1909.	Ditto	— 26th August 1909	— 26th August 1909.
43	3,120—13th August 1909.	Ditto	— 17th August 1909 Strongyloides.	17th August 1909. Trichomonas.
	2,950—16th August 1909	Ditto	— 23rd August 1909 Trichomonas.	+ 23rd August 1909.
	2,960—23rd August 1909.	Ditto	— 26th August 1909	+ 26th August 1909.

Table III—*continued.*

NORMAL MONKEY.

Number of monkey.	Weight (grams).	Stools.	AMŒBÆ.	
			Microscope.	Cultivation.
44	1,490—13th August 1909.	No diarrhœa	— 17th August 1909 Trichomonas.	+ 17th August 1909.
	1,370—16th August 1909.	Ditto	— 23rd August 1909 Trichomonas.	+ 23rd August 1909.
	1,370—23rd August 1909.	Ditto	— 26th August 1909	+ 26th August 1909.
49	1,730—27th August 1909.	Ditto	— 31st August 1909	— 31st August 1909.
	1,710—1st September 1909.	Ditto	— 2nd September 1909	— 2nd September 1909.
	1,690—8th September 1909.	Diarrhœa, no blood or mucus.	— 5th September 1909	+ 5th September 1909.
			— 8th September 1909 —	+ 8th September 1909.
		DIED—9th September 1909.		
52	1,400—27th August 1909.	No diarrhœa	— 1st September 1909	+ 1st September 1909.
	1,340—1st September 1909.	Ditto	— 6th September 1909	+ 6th September 1909.
	1,180—8th September 1909.	Ditto.		
53	2,270—27th August 1909.	Ditto	— 1st September 1909	+ 1st September 1909.
	2,100—1st September 1909.	Ditto	— 6th September 1909	+ 6th September 1909.
	2,070—8th September 1909.	Ditto	— 16th September 1909	+ 16th September 1909.
	2,070—15th September 1909.	Ditto	— 20th September 1909 Trichomonas.	+ 20th September 1909.
	1,920—21st September 1909.	Ditto.		
		DIED—24th September 1909.		
50	2,000—27th August 1909.	No diarrhœa	— 3rd September 1909. Trichomonas.	+ 3rd September 1909.
	1,960—1st September 1909.	Ditto	— 6th September 1909	+ 6th September 1909.
	1,770—8th September 1909.	Ditto	— 15th September 1909	+ 15th September 1909.

Table III—*continued.*

NORMAL MONKEY.

Number of monkey.	Weight (grams).	Stools.	Amœbæ.	
			Microscope.	Cultivation.
	1,700—15th September 1909.	No diarrhœa	— 2nd October 1909	+ 2nd October 1909.
	1,790—21st September 1909.	Ditto	— 13th October 1909	+ 13th October 1909.
	1,800—28th September 1909.	Ditto	— 21st October 1909	+ 21st October 1909.
	1,720—4th October 1909.	Ditto	— 26th October 1909	+ 26th October 1909.
	1,750—12th October 1909.	Ditto	— 3rd November 1909	3rd November 1909.
	1,740—18th October 1909.	Ditto	— 10th November 1909	+ 10th November 1909.
	1,750—25th October 1909.	Ditto.		
	1,750—1st November 1909.	Ditto.		
	1,710—9th November 1909.	Ditto.		
	1,609—16th November 1909.	Ditto.		
	1,660—23rd November 1909.	Ditto.		
51	1,270—27th August 1909.	Ditto	— 1st September 1909	+ 1st September 1909.
	1,230—1st September 1909.	Ditto	— 6th September 1909	— 6th September 1909.
	1,110—8th September 1909.	Ditto	— 16th September 1909	— 16th September 1909.
	1,180—15th September 1909.	Ditto	— 2nd October 1909	— 2nd October 1909.
	1,240—21st September 1909.	Ditto	— 13th October 1909	— 13th October 1909.
	1,240—28th September 1909.	Ditto	— 21st October 1909	— 21st October 1909
	1,220—4th October 1909	Ditto	— 26th October 1909 Trichomonas.	+ 26th October 1909.

Table III—*continued.*

NORMAL MONKEY.

Number of monkey.	Weight (grams).	Stools.	AMŒBÆ.	
			Microscope.	Cultivation.
	1,270—12th October 1909.	No diarrhœa	— 3rd November 1909	+ 3rd November 1909.
	1,280—18th October 1909.	Ditto	— 10th November 1909 Trichomonas.	+ 10th November 1909.
	1,260—25th October 1909.	Ditto.		
	1,250—1st November 1909.	Ditto.		
	1,250—9th November 1909.	Ditto.		
	1,240—16th November 1909.			
	1,200—23rd November 1909.			
54	2,290—27th August 1909.	No diarrhœa	— 31st August 1909	+ 31st August 1909.
	2,060—1st September 1909.	Ditto	— 2nd September 1909	+ 2nd September 1909.
	2,100—8th September 1909.	Ditto	— 15th September 1909	+ 15th September 1909.
	2,070—15th September 1909.	Ditto	— 22nd September 1909 Trichomonas.	+ 22nd September 1909.
	2,030—21st September 1909.	Ditto	— 2nd October 1909	+ 2nd October 1909.
	2,100—28th October 1909.		— 13th October 1909	+ 13th October 1909.
	2,070—4th October 1909.	No diarrhœa	— 19th October 1909 Trichomonas.	+ 19th October 1909.
	2,170—12th October 1909.	Ditto	— 26th October 1909 Trichomonas.	+ 26th October 1909.
	1,880—18th October 1909.	Diarrhœa, no blood mucus.	— 3rd November 1909	— 3rd November 1909.
	1,970—25th October 1909.	No diarrhœa	— 10th November 1909	+ 10th November 1909.
	1,860—1st November 1909.	Ditto.		

Table III—*continued.*

Normal Monkey.

Number of monkey.	Weight (grams).	Stools.	Amœbæ.	
			Microscope.	Cultivation.
	1,710—9th November 1909.	No diarrhœa.		
	1,510—16th November 1909.	Ditto.		
	1,450—23rd November 1909.	Ditto.		
		Died—26th November 1909.		
55	2,230—28th September 1909.	No diarrhœa	— 29th September 1909	— 29th September 1909.
	2,200—4th October 1909.	Ditto	— 5th October 1909	+ 5th October 1909.
	2,370—12th October 1909.	Ditto	— 14th October 1909	— 14th October 1909.
	2,400—18th October 1909.	Ditto	— 20th October 1909	— 20th October 1909.
	2,420—25th October 1909.	Ditto	— 27th October 1909 Strongyloides.	+ 27th October 1909.
	2,380—1st November 1909.	Ditto	— 4th November 1909	— 4th November 1909.
	2,310—9th November 1909.	Ditto	— 13th November 1909	+ 13th November 1909.
	2,300—16th November 1909.	Ditto		
	2,320—23rd November 1909.	Ditto		
56	2,280—28th September 1909.	Ditto	— 29th September 1909	+ 29th September 1909.
	2,310—4th October 1909.	Ditto	— 5th October 1909	+ 5th October 1909.
	2,340—12th October 1909.	Ditto	— 14th November 1909	+ 14th November 1909.
	2,380—18th October 1909.	Ditto	— 20th October 1909	— 20th October 1909.
	2,500—25th October 1909.	Ditto	— 27th October 1910 Trichomonas.	+ 27th October 1909.
	2,530—1st November 1909.	Ditto	— 4th November 1909	— 4th November 1909.

Table III—*continued.*

NORMAL MONKEY.

Number of monkey.	Weight (grams).	Stools.	AMŒBÆ.	
			Microscope.	Cultivation.
	2,540—9th November 1909.	No diarrhœa	— 10th November 1909	— 10th November 1909.
	2,500—16th November 1909.	Ditto.		
	2,510—23rd November 1909.	Ditto.		
57	2,400—28th September 1909.	Ditto	— 29th September 1909	— 29th September 1909.
	2,440—4th October 1909.	Ditto	— 5th October 1909	+ 5th October 1909.
	2,550—12th October 1909.	Ditto	— 14th October 1909	+ 14th October 1909.
	2,610—18th October 1909.	Ditto	— 20th October 1909 Trichomonas.	+ 20th October 1909.
	2,600—25th October 1909.	Ditto	— 27th October 1909	+ 27th October 1909.
	2,630—1st November 1909.	Ditto	— 4th November 1909	— 4th November 1909.
	2,610—9th November 1909.	Ditto	— 13th November 1909	+ 13th November 1909.
	2,510—16th November 1909.	Ditto.		
	2,520— 23rd November 1909.	Ditto.		
58	1,700—28th September 1909.	Ditto	— 29th September 1909	— 29th September 1909.
	1,710—4th October 1909.	Ditto	— 5th October 1909	+ 5th October 1909.
	1,810—12th October 1909.	Ditto	— 14th October 1909 Trichomonas.	+ 14th October 1909.
	1,810—18th October 1909.	Ditto	— 20th October 1909 Trichomonas, strongyloides.	— 20th October 1909.
	1,810— 25th October 1909.	Ditto	— 27th October 1909.	+ 27th October 1909.
	1,790— 1st November 1909.	Ditto	— 4th November 1909	— 4th November 1909.
	1,790—9th November 1909.	Ditto	— 13th November 1909	— 13th November 1909.

Table III—*concluded.*

NORMAL MONKEY.

Number of monkey.	Weight (grams).	Stools.	AMŒBÆ.	
			Microscope.	Cultivation.
	1,800—16th November 1909.	No diarrhœa.		
	1,820—23rd November 1909.	Ditto.		
59	2,300—28th September 1909.	Ditto	— 29th September 1909	— 29th September 1909.
	2,240—4th October 1909.	Ditto	— 5th October 1909	+ 5th October 1909.
	2,360—12th October 1909.	Ditto	— 14th October 1909	— 14th October 1909.
	2,390—18th October 1909.	Ditto	— 20th October 1909	— 20th October 1909.
	2,410—15th October 1909.	Ditto	— 27th October 1909	— 27th October 1909.
	2,490—1st November 1909.	Ditto	— 4th November 1909	— 4th November 1909.
	2,530—1st November 1909.	Ditto	— 13th November 1909	+ 13th November 1909.
	2,580—16th November 1909.	Ditto.		
	2,620—23rd November 1909.	Ditto.		
60	2,280—28th September 1909.	Ditto	— 29th September 1909	+ 29th September 1909.
	2,220—4th October 1909.	Ditto	— 5th October 1909	+ 5th October 1909.
	2,350—12th October 1909.	Ditto	— 14th October 1909	+ 14th October 1909.
	2,330—18th October 1909.	Ditto	— 20th October 1909	+ 20th October 1909.
	2,300—25th October 1909.	Ditto	— 27th October 1909	+ 27th October 1909.
	2,250—1st November 1909.	Ditto	— 4th November 1909	+ 4th November 1909.
	2,440—9th November 1909.	Ditto	— 13th November 1909	+ 13th November 1909.
	2,480—16th November 1909.	Ditto.		
	2,440—23rd November 1909.	Ditto.		

Description of Plates.

The figures in Plates I and II were drawn with a large Zeiss Camera Lucida apparatus, tube of microscope being extended to 160 m.m. Ocular. No. 6. compensating Objective, Zeiss 2 m.m. Apoch. Aperture 1·30. For description see page 13.

Plate I.

Figs. 1, 2, 3 and 4.—A culture of amœbæ from the stool of a case (No. 167) of dysentery. Stained by iron hæmatoxylin method. They show adult amœbæ and contained "merozoits"; also buds which have separated to become young amœbæ. The culture was 24 hours' growth.

Fig. 5.—A culture of amœbæ from the pus of an abscess of the liver, case No. 284. Stained by iron hæmatoxylin method. It shows equal division of nucleus. The culture was three days' growth.

Figs. 6 and 7.—A culture of amœbæ from the pus of an abscess of the liver, case No. 105. They show the nucleus of the amœba with a ring of chromatine, and the cytoplasm contains "merozoits". Fig. 7 shows the differentiation of the cytoplasm into a clear ectoplasm and granular endoplasm. The culture was eight days' growth.

Figs. 8, 9, and 10.—The culture is the same as in figs. 6 and 7. Unstained. They show clearly vacuoles of various sizes.

Figs. 11 and 12.—The culture is the same as in figs. 6 and 7. Stained by iron hæmatoxylin. They show division by budding in amœbæ from liver pus.

Figs. 13, 14 and 15.—The culture is the same as in figs. 6 and 7. Unstained. They show the stage of encystment of amœbæ from liver pus. The various shapes of cysts met with, polygonal and circular, are illustrated.

Figs. 16, 17 and 18.—The culture is the same as in figs. 6 and 7. Stained by iron hæmatoxylin. They show various forms of amœbæ met with in liver pus.

Fig. 19.—A culture from a case of dysentery, No. 117. Unstained. It shows the amœbic cysts from a stool of a case of dysentery. The culture was three days' growth.

Fig. 20.—A culture from liver pus of case No. 193. Stained by iron hæmatoxylin. It shows a rapidly growing culture of amœbæ. The amœbæ are reproducing both by budding and equal division. The culture was four days' growth.

Fig. 21.—The culture is the same as fig. 20. It shows a number of young amœbæ. The young forms favour premature budding.

Plate II.

Fig. 22.—A culture from a stool of a dysentery case No. 203. Stained by Borrel's method. It shows two encysted forms containing many chromatine granules, and two amœbæ which have just escaped from the cyst, the cytoplasm being rich in chromatine and ready to bud rapidly.

Figs. 23 and 24.—A culture from a stool of dysentery, case No. 196. Stained by hæmatoxylin. It shows the occurrence of reproduction by equal division and by budding in the same amœba. Culture was two days' growth.

Fig. 25.—A culture from liver pus of case No. 284. Unstained. Shows various forms and shapes of amœbæ, an ordinary adult amœba, several irregular shaped and encysted forms. The culture was three days' growth.

Fig. 26.—The same culture as fig. 25. Stained by iron hæmatoxylin. It shows division forms from a culture of liver pus.

Fig. 27.—A culture from liver pus. Unstained. It shows the variation in form and size of amœbæ.

Fig. 28.—(Free hand sketch not drawn to scale). Unstained. It shows the phases of the stage of encystment of the amœba. The observations were made on a single amœba growing on agar. (*a*) 12 noon, 15th November 1909. Normal encysted form. (*b*) 2-40 P.M. Bud of protoplasm extruded; movements inside cyst active. (*c*) 2-44 P.M. Larger portion of protoplasm extruded. (*d*) 2-48 P.M. The empty "case" of chitinous material with a small aperture through which the amœba (*e*) has escaped.

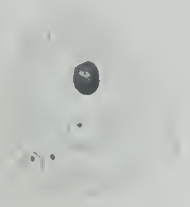
Fig. 29.—A culture from the stools of a case of dysentery. Stained by iron hæmatoxylin. It shows karyokinetic nuclear division.

Plate III.

Fig. 1.—Section of stomach of cat in a case of natural amœbic infection described in the Report, page 22. (A) Depression in mucus coat; the amœbæ are seen to have a dark centre surrounded by a clear peripheral zone. The invasion is early and the amœbæ have not extended beyond the mucus layer. (B), (C), (D), and (E) Mucus, sub-mucus, muscular and serous coats of the stomach. Stained by methylene blue eosine. × (70).

Fig. 2.—A portion of the affected mucus coat of above under a higher power. The amœbæ are seen to be filled with red blood corpuscles, which stain red with eosine; the red cells are contained in the endoplasm, the well defined clear ectoplasm is seen surrounding the endoplasm. The nucleus is placed at the boundary between the ecto—and endoplasm, its chromatine is arranged in a ring at the periphery; a nucleolus is seen in the centre of the nucleus. (× 390).

Fig. 3.—A section of the large intestine of a fatal case of amœbic dysentery in man. An account of the intestinal lesions in dysentery is given in the Report, page 35. The figure shows a portion of the greatly thickened middle coat of the large intestine (the characteristic lesion of amœbic dysentery). In this thickened layer (A) a part of a well circumscribed area of cellular infiltration is seen, and at the wall of this "focus" two typical amœbic forms (B) are observed; these are readily detected by their large size and the characters of the nucleus. (Stained by iron hæmatoxylin and eosine). (× 460).



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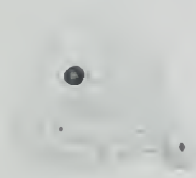

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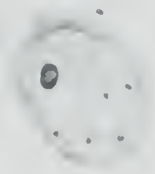

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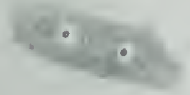

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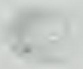

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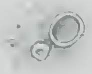

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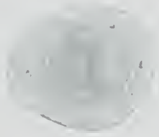

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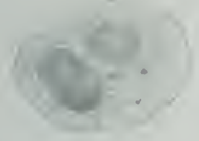

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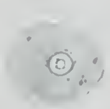

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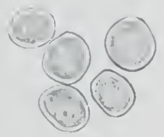

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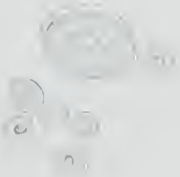

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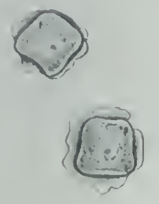

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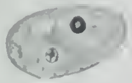

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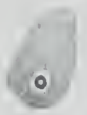

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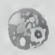

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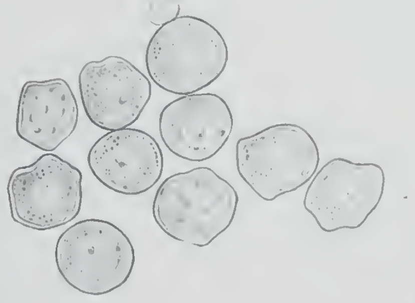

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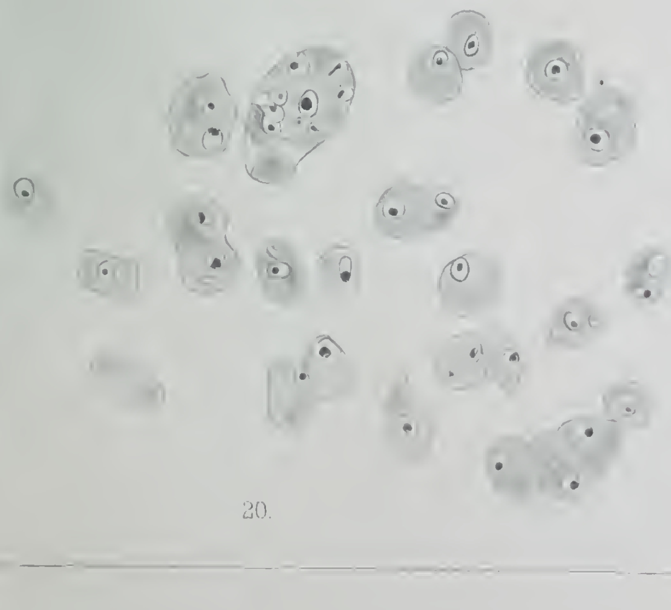

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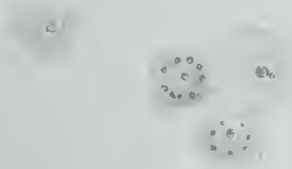

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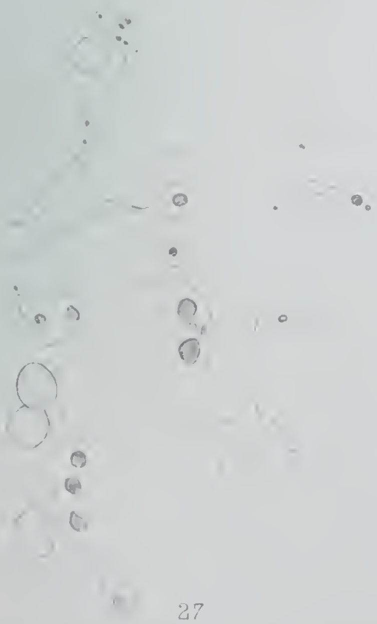

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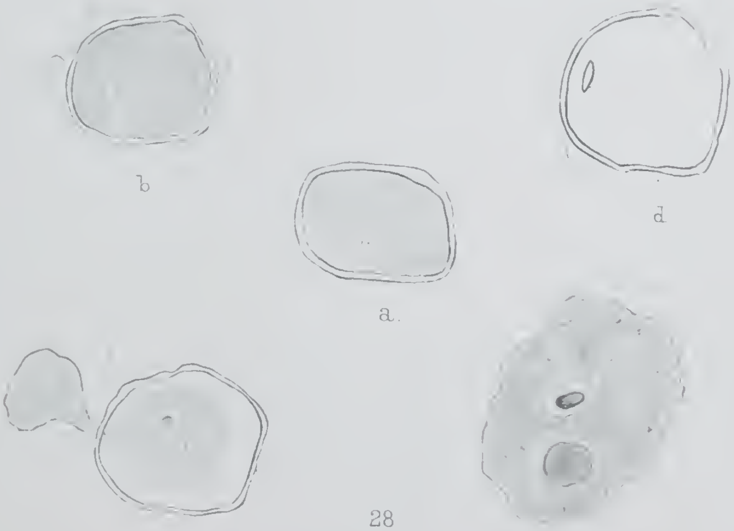

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Fig.1.x70.

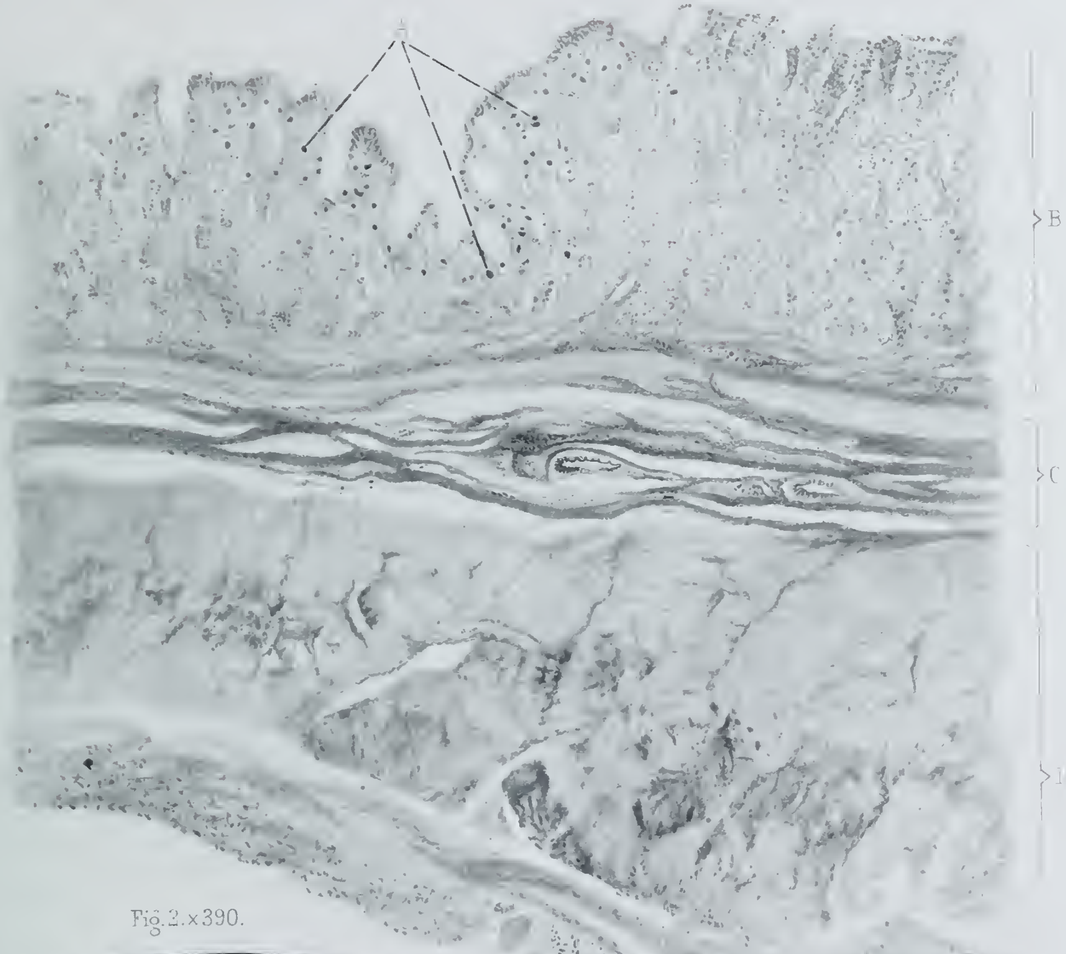


Fig.2.x390.

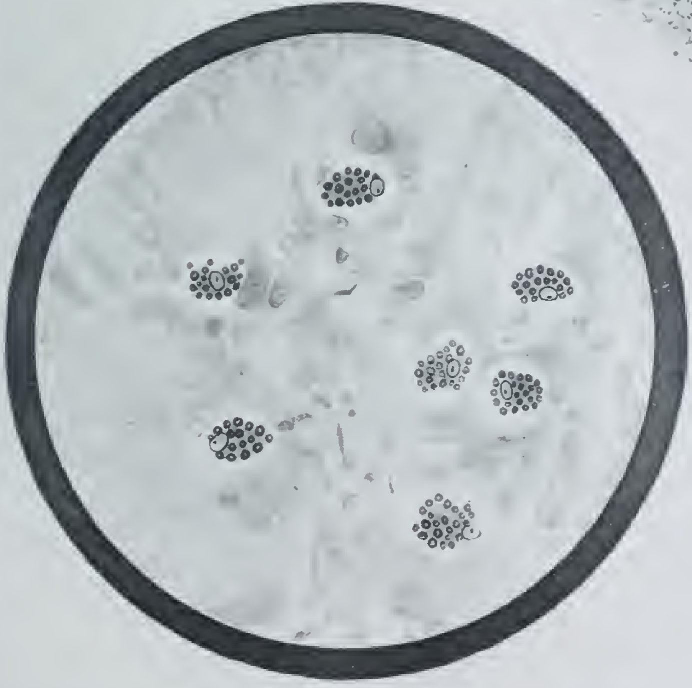
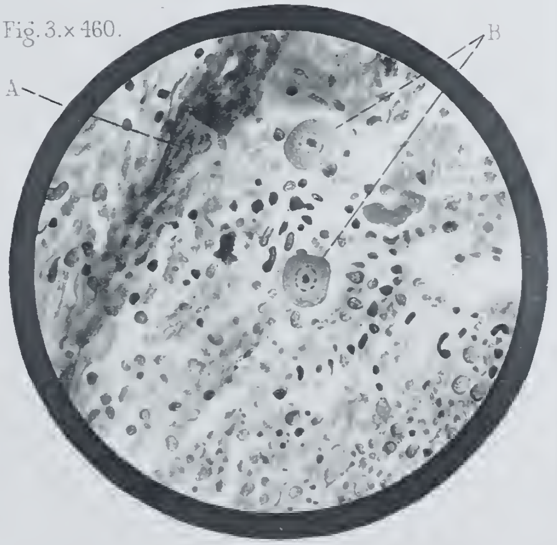


Fig.3.x460.



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ERRATUM.

In Table XIX on page 58 of the Scientific Memoirs, No. 43, under the heading "Site of injection of antitoxin" opposite experiment No. 3 (Control of tetanus culture used) *for* the word "Ditto" *read* "Nil".